ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 62

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Published Quarterly

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ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE . 1010 BEAUMONT BUILDING, 8
BUSINESS OFFICE . P. O. BOX 1345, CENTRAL STATION, 1

ST. LOUIS, MO., U.S.A.

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ANNALS PUBLISHING COMPANY

Annual Subscription in United States and All Foreign Countries is $12.00 in Advance.

610.5
A6
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v.62

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ANNALS
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OTOLOGY, RHINOLOGY
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VOL. 62 MARCH, 1953 No. 1

I

STUDIES ON THE OTIC LABYRINTH

III. ON THE THRESHOLD OF MINIMUM PERCEPTIBLE ANGULAR ACCELERATION

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DULUTH, MINN.

The minimum angular acceleration which is consciously perceptible, presumably due to deviations of the cupula, has been the subject of a number of studies. De Vries[1] studied stimuli applied through very small periods of time (0.1 to 0.25 sec.)—the start, acceleration, declaration and stop all occurring within a fraction of a second and within a turn not exceeding 30°—and concluded that the excitation of the sense cells is a function of time and acceleration. The threshold depends upon the product of time and acceleration if the time is less than one second, but, when the time is longer than one second, this so-called acceleration-time (A.T.) does not hold.

Groen and Jongkees[4] after their studies, concluded that the minimum perceptible angular acceleration for man is about 0.5° per second per second (sec.^{2}). They quote Mach (1875) as having found that 2° per sec.^{2} was the threshold value. Hulk and Jongkees[5]

Work done as visiting research professor in the Department of Otolaryngology, Columbia University, and supported in part by the Research Council of the American Otological Society and the Hayden-Coakley Memorial Fund, and supported in large part by Air Force Grant A.F. 33 (038) 27877.

reported the average as 2.5°/sec.,^{2} with the limits varying between 0.75° and 5.0°/sec.^{2}

The present study has sought to determine the minimum perceptible acceleration and deceleration during sustained turn.

METHODS

The tests in this study were made in an electrically powered Barany turning chair mechanically controlled by two Graham transmissions and so constructed that measured acceleration and deceleration as well as constant rotation at any speed up to 60 revolutions per minute could be produced at will. The tests were made with the subject's eyes closed and his head held at 30° forward inclination. To exclude clues from extraneous light and sound sources, a large hood was lowered over the subject and ear phones were put on him.

The subjects tested were freshmen medical students between the ages of 20 and 23 years.

Two types of tests were employed. In the first, the chair was set turning very slowly (one revolution per minute) and, at a time unknown to the subject, acceleration was begun and increased in 0.25° steps until he responded. Minimal perceptible deceleration was tested in a similar manner, beginning from 9 or 10 r.p.m. In this test there was only a single response for each, acceleration and deceleration, in right turn and in left turn.

In the second test, the subject reported his sensations every 5 or 6 seconds over a period of 10 to 15 minutes, during which time acceleration and deceleration at various rates, as well as constant rates of turn at various speeds, were developed. Acceleration gives a sensation of turn in the direction in which the chair is turning, whereas deceleration gives a sensation of turn in the opposite direction, while, theoretically, a constant rate of revolution should give no sensation of turn. The subjects were instructed to report "right" or "left" only when they felt certain of a sensation of turn, otherwise they were to report "still" (no acceleration or deceleration).

Calibrating measurements were made upon the chair when the tests were finished. These measurements indicated a fairly high degree of accuracy for all accelerations up to 4° per sec.,^{2} excepting 0.25,° which last was not accurate. Three different rates of acceleration and deceleration were used: 0.5° per sec,^{2} 0.75° per sec.^{2} and 1.0° per sec.^{2} They were used interchangeably in a random manner together with "still" (constant rate of turn) in order to avoid any pattern which might give the subject a clue as to what to expect.

TABLE I.

SHORT TEST.

SUBJECT	RT. AC.	RT. DC.	L. AC.	L. DC.
A. F.*	1.0°	1.5°	1.5°	0.75°
T. A.	0.5	0.75	0.75	0.75
N. C.	0.75	0.5	0.5 1.0	0.5
M. W.	0.75 0.75	0.5 0.5	1.25 1.0 0.75	0.5 0.5
J. S.	0.75 1.5 1.5	1.0	1.25	1.0
J. Z.	0.5 1.0	0.5 1.0	1.0	2.0
S. O.	1.0	0.75	1.0	0.75
A. M.	1.25 1.50	0.5	1.5	0.5
J. S.	0.75	0.75	1.0	0.5
D. P.	1.0	1.0	0.75	0.5
R. R.	1.0 0.75	0.75 0.75	0.75 0.75 0.75	0.5** 0.5 0.75
R. S.	1.0	0.75 0.5	0.5	0.75
D. R.	1.0 1.0	0.5	0.75	0.5
R. S.	0.62 1.0 0.5	2.25	0.87 1.0	1.5
T. M.	1.75 1.25	1.0	1.5 1.5	1.0 1.25
D. L.	1.0 0.5	1.0	1.25 1.0	0.5 0.75
A. M.	0.75	0.75	0.75	0.5

*In one test, this subject called right acceleration "left" up to 3.25.°

**Failed to report one deceleration up to 1.25.° Others seemed accurate.

TABLE I.—(Continued).

SUBJECT	RT. AC.	RT. DC.	L. AC.	L. DC.
B. P.	0.75	3.0	2.0	1.0
	0.75		2.12	
	1.0			
J. S.	1.5	1.5	1.25	0.75
			1.50	0.25
J. W.	0.75	0.75	1.0	0.75
			0.75	1.25
			1.75	
P.	0.75	1.0	0.75	0.75

Acceleration and deceleration per second per second in degrees as sensed by 21 different individuals in the first test. Rt. ac.—right acceleration. Rt. dc.—right deceleration. L. ac. —left acceleration. L. dc.—left deceleration.

FINDINGS

First Test: The first test (increasing acceleration or deceleration steadily until the subject responded) was made on 21 different subjects. Acceleration and deceleration were increased in steps of 0.25° per sec.^{2} The results are shown in Table I. Acceleration and deceleration were sensed about equally. The threshold varied somewhat with most subjects detecting 0.75° or 1.0° per sec.;^{2} a few seemed to detect 0.5° per sec.^{2} regularly, while some were uncertain up to 2° or 3.° The extremes in this group varied from 0.25° to 3° per sec.^{2}

From this test, the examiner received the impression that the responses were not very dependable; upon repetition of the test, the results were not always consistent.

Second Test: The second form of test (the subject was kept turning slowly and continuously while subjected to minimum angular acceleration and deceleration) was made on 26 subjects. Another group of about the same size had been tested previously, but, owing to the fact that the mechanism of the chair functioned faultily during deceleration, these figures were discarded and the chair was rebuilt before the tests here reported were made.

When put into statistical form, the responses in the second test were found to be surprisingly uniform and seemed to follow a pattern.

TABLE II.

ACCELERATION AND DECELERATION AT 0.5°/sec.²

			INCORRECT	
		COR.	OP.	STL.
210	R. ac.	66%	10%	23.3%
124	R. dc.	51	6.4	41
214	L. ac.	61	5.6	33
131	L. dc.	67	5	
424	Acceleration	63.6	7.7	28.5
255	Deceleration	60	5.5	34.5
679	Total	62.7	7	30.5

Summary of results of acceleration and deceleration at 0.5° per second squared, in 679 responses. Responses were correct in 62.7%, 7% were incorrectly designated as a turn in the opposite direction, while 30.5% were incorrectly interpreted as 0 acceleration or deceleration. Cor.—correctly designated. Op.—incorrectly designated as being the opposite direction. Stl.—incorrectly designated as no acceleration or deceleration.

At an acceleration of 0.5° per sec.,² about 64 per cent of the responses were correct; in deceleration about 60 per cent were correctly called. The accuracy for acceleration and deceleration combined was about 63 per cent (Table II). Thirty seven per cent were incorrect but only 7 per cent were called in the direction opposite to that of the turn; the other 30 per cent were incorrectly given as "still" (no acceleration or deceleration).

The responses to 0.75° per sec.² were much the same: 63.1 per cent correct and 37.1 per cent incorrect. Eight plus per cent were designated incorrectly as the opposite direction of turn and 28.8 per cent were given incorrectly as "still" (Table III).

At 1.0° per sec.² the accuracy was higher with almost 70 per cent correct and only 30 per cent incorrect. Only 5.5 per cent were designated in the opposite direction of turn and 24.5 per cent as "still" (Table IV).

At all three of these acceleration rates, the responses were roughly two-thirds correct. About 25 to 30 per cent were incorrectly designated as "still" (no turn) and only between 5 and 10 per cent gave the opposite direction of turn.

TABLE III.

ACCELERATION AND DECELERATION AT 0.75/sec.^{2}

		COR.	INCORRECT OP.	STL.
194	R. ac.	66%	11.3%	22.6%
136	R. dc.	61.8	8.1	30
185	L. ac.	66	10.8	23.2
160	L. dc.	57.5	2	40.6
379	Acceleration	66	11	23
296	Deceleration	59.5	4.7	35.8
675	Total	63.1	8.3	28.8

Summary of results of acceleration and deceleration at the rate of 0.75° per second,^{2} in 675 responses. Responses were correct in 63.1%, 8.3% were incorrectly designated as a turn in the opposite direction, while 28.8% were incorrectly interpreted as 0 acceleration or deceleration.

The threshold at which angular acceleration is sensed does not seem to be as definite, for instance, as that for hearing—and this latter is not especially sharply defined. There seem to be wide individual variations, as seen in the case of N. C., who was so inaccurate that no determination of deceleration could be made. Each time acceleration was terminated, he reported deceleration (turn in the opposite direction) altho the r.p.m. were held constant. In one test he persistently reported right turn although he was accelerated to the left 1.5° per sec.^{2} up to 14 r.p.m. On the other hand, A. M. sensed a change of 0.5° per sec.^{2} without fail, whether from "still" (even rotation) to either acceleration or deceleration or vice versa. He was usually, but not always accurate in his designation, but he sensed the change. B. P. was uncertain up to 3° per sec.^{2}

The time element is a large factor. There is a definite latent period of a few seconds following each change at these low rates. Not only is there a lag in recognition of change, but if an acceleration which is barely sensed is maintained for some little time, the sense of turn is lost. Fatigue may be a factor. Despite efforts to expedite the examinations, most subjects were under test (with periods of rest) for upward of two hours.

TABLE IV.

ACCELERATION AND DECELERATION AT 1.0°/sec.^{2}

			INCORRECT	
		COR.	OP.	STL.
77	R. ac.	74%	4%	22%
95	R. dc.	63.1	2	34.7
77	L. ac.	79.2	8	13
57	L. dc.	63.1	10	26.2
154	Acceleration	76.6	5.8	17.5
152	Deceleration	63	5.5	31.5
306	Total	70	5.5	24.5

Summary of results of acceleration and deceleration at the rate of 1.0° per second,^{2} in 306 responses. Responses were correct in 70%, 5.5% were incorrectly designated as a turn in the opposite direction, while 24.5% were incorrectly interpreted as 0 acceleration or deceleration.

Table V, analyzing 326 responses during slow constant turn at the beginning of the tests before any acceleration, reveals that 86 per cent were accurate, the subjects correctly reporting "still."

Each subject was carefully instructed that he should not report "turn" unless he felt sure of it. If there were doubt in his mind, he was asked to report "still" (no acceleration or deceleration). Despite this warning, the responses to constant turn, subsequent to the initial control test, were highly inaccurate, i.e., if the subject had sensed acceleration or deceleration, he seemed much less able to determine constant turn ("still"). In 1361 responses (Table VI) only 52 per cent were correctly given as "still"; about 24 per cent were reported "right turn" and another 24 per cent "left turn." The inaccuracy in this test was the greatest found. The responses followed a random pattern.

After they had been subjected to minimal acceleration and deceleration, these intelligent subjects, who were told not to report turn unless they were sure of it, reported a sense of turn in 48 per cent of 1364 instances when there should have been none.

The writer has become convinced that there is such a thing as gyratory illusion, corresponding to the well known optical illusions (diagrams which may be visually interpreted in either one of two

TABLE V.

CONSTANT SLOW RATE OF TURN.

INITIAL ACCELERATION—0.

Total responses	326	
Correctly called	278	85.3%
Incorrectly called:		
Right	27	8.4%
Left	21	6.4%

Summary of 326 responses to the control tests of 0 acceleration, made at the beginning of the tests before there had been any acceleration or deceleration. These control tests were made at a constant slow rate of turn, about 1 RPM. Eighty-five per cent were correctly called 0 acceleration, about 15% were incorrectly called acceleration or deceleration, either right or left.

ways). When the subject reports right or left turn and there actually is no angular acceleration, he may, none the less, be sensing it distinctly in consciousness and not simply be uncertain as one is at the threshold of hearing. Optical illusions seem to be produced in consciousness from ambiguous or unusual retinal signals. Similarly, minimal, ambiguous signals from the labyrinth seem to develop in consciousness a definite sense of turn. This "gyratory illusion," if such it is, changes with facility and quickly from right turn to left turn and vice versa.

SUMMARY

(1) Using angular acceleration of a Barany chair, mechanically controlled by two Graham transmissions, minimum perceptible angular acceleration was studied in two ways. First, starting with one turn per minute, by increasing the acceleration (or deceleration from a period of acceleration) until the subject correctly reported a turning sensation. Using this method, the threshold varied between 0.25° per sec.^{2} and 3.0° per sec.^{2} However, threshold values of either 0.75° or 1.0° per sec.^{2} were found in 17 of 21 subjects tested in this way. It was felt that this test involving a single judgment for acceleration and another value for deceleration was not very dependable. In the second type of test the subject was kept turning continuously while subjected to various changes in angular acceleration and deceleration.

TABLE VI.

CONSTANT RATE OF TURN AFTER ACCELERATION AND/OR DECELERATION HAD BEEN TESTED.

ACCELERATION (SUBSEQUENT)—0.

Total responses	1361	
Correctly called	718	52.7%
Incorrectly called:		
Right	318	23.3%
Left	325	23.9%

Summary of 1361 responses to 0 acceleration after the subject had experienced acceleration or deceleration or both. The pattern of response is almost a mathematical random: 52% were correctly called and 48% were incorrectly called. Of those incorrectly called there were about an equal number of right and left.

(2) An angular change of 1.0° per sec.^{2} was sensed correctly in 70 per cent of determinations by young men between 20 and 23 years of age; 0.5° per sec.^{2} and 0.75° per sec.^{2} were only slightly less accurate. Although about 30 per cent were inaccurately designated, only between 5 and 10 per cent were given as being in the opposite direction of turn. The rest were incorrectly designated as "still."

In the initial control test, with the chair turning very slowly, before any acceleration or deceleration had been used, 86 per cent of the responses were correctly given as "still." Subsequently, in the testing, as the chair was brought into a constant rate of turn and the subjects should have reported "still," the responses were so inaccurate as to make practically a random pattern. Only 52 per cent of 1361 responses were correctly called "still," about 24 per cent were called "right" and 24 per cent "left."

This is perhaps the most important finding in this study. When a subject is turning slowly without acceleration or deceleration, after having been subjected to low rates of both, he seems to be unable to determine when the acceleration or deceleration has ceased. Although these young men were told to report "still" unless they were sure of a right or left turn, almost half of the responses reported right or left turn when there was none.

(3) Near threshold a "gyratory illusion" similar to the optical illusions from ambiguous or unusual retinal signals seems to occur. Signals from the labyrinth similar in their ambiguousness seem to

develop in the consciousness a definite sense of turning in one direction or another, even when there is no change in acceleration.

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II

INTERCILIARY STRUCTURES IN THE EPITHELIUM OF THE UPPER PART OF THE RESPIRATORY TRACT

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In a recent study on the vibratile cilia of the tracheal cells, one of us[3] published a number of electron micrographs showing the structure of these cilia. In some of the pictures, a number of thin, finger-like processes 1-2 μ long, were seen to protrude from the cell surface between the cilia.

We have now studied these processes in a large number of specimens from different mammals. We have found them to be fairly constantly present, even after the use of various fixatives (10 per cent formol, osmic acid and a veronal-buffered osmic acid solution according to Palade.)

When using these fixatives, cilia of a fairly uniform appearance were observed in the trachea or nasal mucosa of the guinea-pig, rabbit, dog, cow and man. The vibratile part of these cilia consisted of a fibrillar core and a peripheral sheath as described earlier.[5] A varying number of fine, finger-like protoplasmic processes with an exceedingly thin outer membrane (Figs. 1 and 3) were almost always found between the cilia. Their length was about one-fifth of that of the vibratile cilia. They bore no resemblance to the cilia, but

Fig. 1.—Cilia from the trachea of a dog. The fibrillar, denser, axial core and the thin peripheral sheath can be seen in the long cilia. At the lower end the interciliary processes are evident. Osmic acid fixation. Magnification about 10,000 x.

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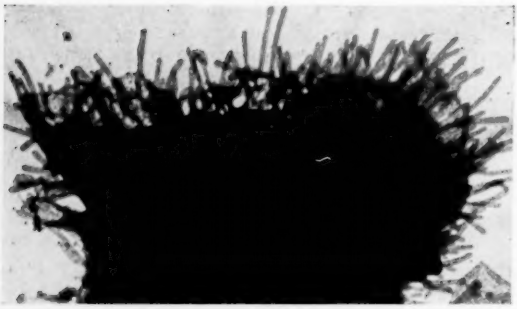

Fig. 2.—Epithelial surface from the olfactory region in man. A large number of thin, finger-like processes are seen. These structures are very numerous in the olfactory region (cf., papers by Bloom and Engström, Engström and Bloom). There is a great resemblance between these structures and the interciliary processes in the epithelium of the upper part of the respiratory tract. The former structures are considered by us to be of great importance for the understanding of olfactory physiology. Osmic acid fixation. Magnification 6,500 x.

consisted merely of protoplasmic processes of somewhat uniform structure. They had no basal bodies and were not divided into a central core and an outer sheath as are the cilia. They were not distinctly differentiated from the cellular protoplasm and thus closely resembled the striated border of the intestinal mucosal cells. The relationship between the cuticle and the protoplasmic processes could not be clearly seen in our preparations and a further study will be made on sections. When using osmic acid fixatives, the processes showed slight surface granulation.

There was a striking similarity between these protoplasmic processes and the finger-like protrusions found in the olfactory region by Bloom[1] and Bloom and Engström.[2, 4]

A problem of considerable importance is whether these processes may have some kind of resorptive function. The first impression was that the interciliary processes comprised some form of undeveloped or potential preciliary structures. No proof of this has, however, been forthcoming. No intermediate stages in ciliary formation have hitherto been observed.

SUMMARY

In a study on the structure of vibratile cilia in the respiratory tract, basal, interciliary, protoplasmic processes have been observed.

These processes are of fairly uniform length in each preparation, but vary in both number and length in different species. They dif-

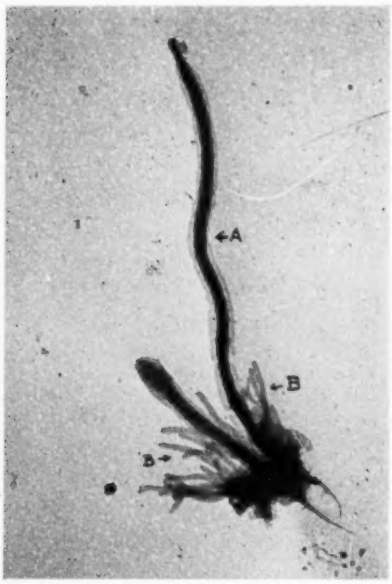

Fig. 3.—Tracheal cilia from a dog. Osmic acid fixation. A—cilium, B—interciliary processes. Magnification about 13,000 x.

fer distinctly from the vibratile cilia, but strongly resemble the striated border of the intestinal cells and the finger-like protrusions found in the olfactory region and described by Bloom and Bloom and Engström.

Their possible importance for resorption or ciliary regeneration is discussed. Several electron micrographs are presented.

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III

FURTHER MICROSCOPIC STUDIES OF THE SECRETIONS OF THE EXTERNAL AUDITORY CANAL

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AND

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The increased attention focused on inflammations of the external auditory canal indicates the need for a simple and rapid method of diagnosis, prognosis and treatment of the various diseases of the ear. It is generally agreed that a careful study of the cellular elements of the external ear aids materially in the differential diagnosis of diseases affecting the middle and external ear.

We have attempted to show by examining pre-treatment smears and then "serial slides" of the external ear secretions that a differential diagnosis may be made readily, and finally that the course of the disease and the response to treatment may be followed.

Although there are many reports of bacteriological studies of the secretions of the external auditory canal, only an occasional reference to the cytology of these same secretions has been found.

Politzer[1] noted large numbers of "micro-cocci" present in the scales of acute diffuse external otitis. Dean and Pfingsten[2] examined the secretions in two cases of external otitis and reported finding epithelial cells and bacteria. Senturia, Adler and Matthews[3] studied the cytology of the secretions of 125 cases of external otitis and reported that "except in complicated cases (other than furunculosis) epithelial cells were present with an absence of leucocytes." They concluded that "the presence of neutrophiles, lymphocytes and mucus strands in secretions taken from the external auditory canal suggests a diagnosis of middle ear disease, circumscribed or complicated diffuse external otitis." Conley[4] reported 32 cases of external otitis in which he observed epithelial cells, leucocytes, red blood cells, bacteria and fungi in the secretions from the canal. Unfortunately, he did not define the specific type of cases. Senturia[5] in a report on

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diffuse external otitis mentioned that myriads of bacteria and epithelial cells were to be found. Senturia and Marcus[6] pointed out the cytologic findings in two categories of external otitis.

After an extensive study of the secretions obtained in 262 ears with otitis media and mastoiditis, Koch[7] described 52 ears in which eosinophilic cells were observed in the secretions of the middle ear. He quotes Dohlman as finding "a large amount of eosinophilic leucocytes in the secretions." More recently Kuhn[8] stated that eosinophiles are frequently present in the secretions of chronic otorrhea.

Proetz[9] in the examination of the secretions of an allergic middle ear noted cocci, leucocytes, epithelial cells and an abscence of eosinophiles. Dean[10] stated that in otitis media after myringotomy, one finds many leucocytes but that not many eosinophiles are to be noted in the discharge. Dean and Pfingsten[2] stated that neutrophiles were present in the secretions of twenty-six cases of chronic otitis media and in nine cases of acute otitis media. Friel[11] stated that in the discharge of chronic otitis media red blood cells, white blood cells and numerous microorganisms such as the streptococci or pneumococci are to be found.

Blank et al[12] studied the cytology of the fluid exudate of herpes simplex, herpes zoster and varicella (not from lesions of the external ear, however) and described the identifying multinucleated epithelial cells seen in the fluid. Bryan and Bryan[13] report characteristic changes in the cellular elements of nasal secretions during virus upper respiratory infections.

Some reports dealing with the pH changes of the external auditory canal have appeared.[14-17] Most authors agree that the pH of the external auditory canal is normally slightly acid while that of the diseased canal is more alkaline. It is conceivable that the pH of the external canal may have some effect on the number of leucocytes seen there. Although dealing with pleural exudates, Menkin[18] stated that the polymorphonuclear cells are incapable of surviving in an acid pH and that there is a definite cause and effect in the pH of the medium and the cytology of the pleural exudate. He demonstrated a drop in the percentage of leucocytes during the fall of the pH from the alkaline to the acid side. It is remotely possible that Menkin's findings could well apply to the exudate found in the external auditory canal.

METHODS

The ear canals were cleansed of exudate either with a cotton swab or by irrigating the canal with a 3 per cent hypertonic solu-

tion of sodium chloride. Two smears were made of the exudate remaining in the region of the sulcus of the canal. In the cases of furunculosis, the material for smears was taken either directly from the site of incision or from the point of spontaneous drainage. The secretion was transferred to the slides by rolling the applicator across the surface of the slides or by gently streaking the surface of the slides with the swabs. The former method is preferred since it results in less destruction and distortion of the cellular elements present in the secretions and makes for a more even distribution of these elements on the slide. The slides were allowed to dry and then were stained as soon as possible.[19] One was stained with Wright's stain for cellular identification and the detection of mucus; the other was flamed and then stained with Gram's stain for the differentiation of bacteria. In those cases where the clinical symptoms indicated a possibility of fungus infection of the canal, direct wet preparations were made of the exudate using Lactophenol Cotton Blue as a mounting medium.[20] A Gram's stain and a Wright's stain were also made on these cases.

When possible, smears were taken before, during and after treatment. Very often daily smears were made in order to observe any cytologic changes which occurred after treatment had been initiated. In order to evaluate the findings on the direct smears, frequent concomitant cultures of the external auditory canals were taken.

The cases were divided into categories according to the following modified classification.[6]

A. Normal
B. External Otitis
    1. Acute Diffuse
        a. Uncomplicated
        b. Complicated
    2. Chronic Diffuse
    3. Neurogenic Dermatitis
    4. Infantile Dermatitis
    5. Furunculosis
    6. Otomycosis
    7. Myringitis Bullosa
C. Otitis Media
D. Postoperative Fenestration and Mastoiditis

Smears of the ear discharge were examined and the quantity of the above elements were evaluated from one to ten plus. Thus

TABLE 1.

SHOWING THE NUMBER OF PATIENTS, NUMBER OF EARS AND THE NUMBER OF SMEARS EXAMINED IN ALL CATEGORIES OF EXTERNAL OTITIS.

CATEGORY	NUMBER OF PATIENTS	NUMBER OF EARS	NUMBER OF SMEARS
Normal	9	16	16
Acute Diffuse*	61	78	218
Chronic Diffuse	11	16	36
Furunculosis	7	7	11
Infantile Dermatitis	3	5	14
Neurogenic Type	25	38	85
Otomycosis	13	16	40
Otitis Media	13	13	36
Postoperative Fenestration and Mastoidectomy	11	11	57
Total	153	200	513

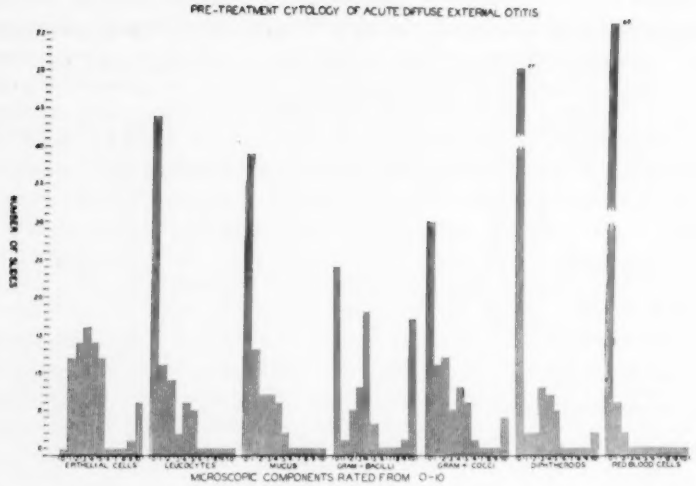
*Includes both the uncomplicated and complicated types.

a rare to an occasional element was considered as a one plus and myriads of cells were reported as ten plus. The occurrence of the following elements was reported:

1. Epithelial cells
2. Leucocytes
3. Eosinophiles
4. Lymphocytes
5. Erythrocytes
6. Mucus
7. Bacteria
8. Fungi and Spores

RESULTS

Table 1 shows the number of patients, the number of ears and the total number of smears studied in this series. Smears were obtained before, during and at the conclusion of therapy. In some cases



Graph 1.

it was possible to obtain only the pre-treatment smears. In other cases regular smears were observed throughout the course of treatment.

One-hundred-and-fifty-three patients with 200 involved ears were studied. A total of 513 smears were examined; in some cases only a single diagnostic pre-treatment smear was available while as many as 15 smears were studied in selected cases.

Graph 1 shows the distribution of the various microscopic elements as found in the pre-treatment smears of all the complicated and uncomplicated cases of acute diffuse external otitis. It may be observed that in any given case, a wide distribution of cellular elements may be found but that a characteristic picture evolves, as will be discussed later.

The findings on the smears during and following treatment could not be charted easily. In general the following sequence of events took place. In a majority of the cases where leucocytes were present on the initial smear, an increase in their number occurred after therapy was started. The number of these cells declined during the course of treatment to a negative reading on the final smear. It is noteworthy that the number of lymphocytes increased as the clinical improvement occurred. No significant increase in eosinophiles was apparent during convalescence.

In most of the smears observed, there was a striking decrease in the number of bacteria during therapy. The final smear would often show only an infrequent organism, usually a gram-positive coccus or a spore-bearing rod. Occasional exceptions occurred where, despite striking clinical improvement, the smear showed myriads of bacteria. In all categories, mucus promptly decreased as soon as therapy was begun.

COMMENT

A differential diagnosis of external otitis can frequently be made by a study of the secretions from the diseased external auditory canals. It is necessary for the clinician to acquaint himself with the cellular elements which are present in normal and pathological secretions so that he may recognize and interpret the significance of these elements. Once the clinician becomes familiar with the morphology of the various diseases and the cellular components found in the secretions of such diseases, a more rapid means of diagnosis and treatment will become available to him.

It is our impression that there is a characteristic picture to be found in the secretions of most of the categories of disease affecting the external canal. There is a very strong similarity in the cellular elements of all types when seen on smear, yet to one who studies these secretions carefully, it is apparent that each type has many of its own characteristics.

In our series we have found the cytology of the normal ear to be one of epithelial cells in small numbers, gram-positive cocci and a few diphtheroids. Our confirming cultures have regularly shown a scanty growth of staphylococci and diphtheroids.

As a result of the comparison of the clinical and microscopic findings in acute diffuse external otitis, it is suggested that a division into uncomplicated and complicated sub-types be made.

In uncomplicated acute diffuse external otitis, no leucocytes are to be seen. The predominant organism is a gram-negative bacillus although one may find a large number of gram-positive organisms also. Mucus may be present but not in all cases. Non-nucleated epithelial cells usually occur in profusion.

In complicated acute diffuse external otitis, neutrophiles are almost always present* with an occasional eosinophile and lymphocyte

*Preliminary pH studies in these cases show a pH range of 6.0-7.0. When compared to our control series, this suggests a small but definite shift towards the alkaline side. In this regard it may be well to keep Menkin's theories in mind.

to be found. The bacterial flora includes myriads of gram-negative bacilli and many gram-positive organisms (usually cocci). One often finds the thick, evenly staining, typically grouped diphtheroid organisms. Mucus is always seen in complicated acute diffuse external otitis, usually in large amounts. Red blood cells are to be noted in many cases. Epithelial cells are present in varying number and it is noteworthy that many of them are of the nucleated type.

The chronic diffuse type of external otitis presents a similar cytologic picture to that seen in complicated acute diffuse external otitis. Leucocytes and red blood cells are seen in greater quantity. The bacterial flora includes myriads of gram-negative bacilli and many gram-positive cocci with the former predominant. Mucus strands are in abundance and a lessening of the epithelial elements frequently occurs.

In furunculosis there is usually a large number of leucocytes and red blood cells in the secretion, especially where spontaneous drainage has occurred. The bacterial flora is primarily one of a gram-positive organism (usually a Staphylococcus) with rarely a commensal gram-negative organism in small numbers present. A goodly amount of mucus is present and no eosinophiles or lymphocytes are to be seen in the initial phase of the infection. The presence of the gram-negative organisms is believed to be the result of maceration of the canal with secondary infection.

Infantile dermatitis appears as a classical cytologic picture. Myriads of short, thick, evenly staining, gram-positive bacilli, typical of the diphtheroids are to be found. Very often the club-shaped, involution forms of this organism are to be seen. A few gram-positive cocci may be visualized as well as many epithelial cells.

In the neurogenic category, fewer epithelial cells are observed on the smear than in any other type excepting the normal ear. The predominant organism is usually a gram-positive coccus (Staphylococcus) and gram-negative organisms are seldom seen. When they occur, however, they are present in the same number and possibly for the same reason as in furunculosis. A rare neutrophile is present and never any eosinophiles or lymphocytes. Occasionally, red blood cells are found, particularly in those cases where "fissuring" has occurred.

In otomycosis the identifying feature is the finding of mycelial elements (usually quite pronounced, although sometimes one must make a thorough search for them) and spores. Many neutrophiles are to be seen. Non-nucleated epithelial cells are increased. Mucus, red blood cells and lymphocytes remain about the same as

those observed in complicated acute diffuse external otitis. Eosinophiles are increased in this type of disease.

In chronic otitis media there is a striking increase in the number of neutrophiles and red blood cells along with a marked increase in all elements. Bacteria are present in overwhelming numbers, sometimes in such profusion as to make differentiation between the organisms impossible. Tremendous numbers of gram-positive and gram-negative organisms are present in both smear and culture.

When smears were obtained from postoperative fenestration and mastoidectomy cavities, neutrophiles were present in large numbers although not as great as in otitis media. We have found more eosinophiles present in the secretions of the postoperative cases than in any of the other categories. Gram-positive organisms (cocci) are seen in the majority of the cases. Fungi are frequently cultured from these cavities.

A few cases of herpetic infections of the external auditory canal have been observed. Fluid taken directly from the blister was shown to contain large epithelial cells with two or more nuclei, somewhat similar to that described by Blank.[12] In the large mononuclear cells (when stained with Giemsa's stain) magenta colored inclusion bodies were observed in the cytoplasm of the cells. Neutrophiles and mucus were found in the fluid but no bacteria were visualized. It is not believed that these findings are in themselves diagnostic of a herpetic infection of the canal, but they are suggestive and will be studied.

CONCLUSIONS

1. The use of a simple yet rapid cytologic method for the diagnosis and prognosis of disease of the external and middle ear is recommended.

2. It is suggested, on the basis of the microscopic findings and for the purpose of classification and treatment, that the disease entity referred to as acute diffuse external otitis be divided into complicated and uncomplicated sub-types.

3. An analysis of the elements found in the secretions obtained from the external auditory canal demonstrates that each category of disease has some of its own characteristic cytologic findings.

4. Differentiation of middle and external ear infections is frequently possible by an examination of the microscopic components of the ear secretions.

5. Further studies of the cytologic changes in the various categories of external otitis, otitis media and mastoiditis should make possible more accurate diagnosis and prognosis.

This study was supported by funds provided under contract AF 33(038)28643 with the USAF School of Aviation Medicine, Randolph Field, Texas.

Appreciation is expressed to Colonel Paul A. Campbell, Lieutenant Colonel James E. Lett, Verna Alford, and Nancy Hellman for assistance in the preparation of this work.

EUCLID AVENUE AND KINGSHIGHWAY BLVD.

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IV

OTOGENIC INTRACRANIAL COMPLICATIONS IN THE "ANTIBIOTIC ERA"

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Otologists are generally agreed that while the systemic effects of chronic ear disease are benefited by antibiotics, they have no curative effect on either bone necrosis or cholesteatoma. These conditions require surgical eradication urgently if intracranial complications develop. In the management of acute otitis media such agreement does not prevail.

The literaure of the past fifteen years offers sufficient evidence of the miraculous cures of otogenic complications with antimicrobial and chemotherapeutic measures. As a result an unwarranted sense of security has developed in the public to the degree that undue pressure is placed on the otologist who advises surgical treatment.

The purpose of this presentation is to demonstrate that not all intracranial complications of otogenic origin are prevented or respond to antibiotic therapy, and secondly, that when they do not effect immediate resolution of the disease, mastoid surgery is indicated. When this surgery is performed early enough and thoroughly, these complications can be cured. When surgery is delayed or is inadequately performed, the morbidity is increased, serious sequelae and mortalities result.

The following cases were chosen from among many seen at the Illinois Eye and Ear Infirmary in recent years. It is true that such cases are often referred to this type of institution when complications ensue. However, the following cases do illustrate intracranial complications developing under extensive antibiotic therapy.

CASE 1.—*Otitis media, subacute, pachymeningitis externa and meningismus.* C. J., white female aged 29, was referred with the following history: On August 28, 1950, the patient developed an acute otitis media, left, which perforated spontaneously the follow-

ing day. She was immediately placed on penicillin procaine, 300,000 units daily intramuscularly, and chloromycetin, 2000 milligrams daily, and triple sulfa's, one gram four times a day. This therapy was continued for 20 days, during which the discharge gradually lessened and the patient was allowed to return to work on chloromycetin therapy. One day later the patient's temperature went up to 103° with chills, headache and stiff neck. Meningitis was suspected and a spinal tap was done. This showed an increased cell count, but no bacteria were present. She was again placed on penicillin therapy, and streptomycin, 1 gram daily, and terramycin, 2000 milligrams daily, were added. On the following day, September 26, 1950, a mastoidectomy was performed. The surgeon who operated described the pathology as follows: "When I removed the mastoid cortex I felt ashamed that I had operated. The trabeculae, while soft, were intact. The mucosa was slightly thickened, grayish, and a few cells were filled with a thin secretion. I did an antrotomy and quit." Following the operation the patient's drainage became profuse, but she felt better and the temperature receded. Postoperatively while in the hospital the mastoid wound was irrigated with tyrothricin solution daily. Again the discharge lessened, but with this a sense of pressure developed and headaches and lowgrade, constant fever continued. At this point, on October 10, a neurosurgical consultant was called to rule out a brain abscess. A second spinal tap was performed and the fluid was normal except for a slightly increased cell count. He suggested further mastoid surgery. Throughout this period the patient was on chloromycetin, penicillin and terramycin. On November 7, 1950, a second mastoid operation was performed.

The report on this operation was given as a thorough mastoidectomy. Postoperatively the patient again had profuse drainage and relief from symptoms. During the last two weeks in December, 1950, the left ear became dry for two weeks. Early in January, 1951, profuse drainage, headaches and constant, lowgrade fever recurred. Throughout this time she was on chloromycetin and terramycin and periods of penicillin therapy. The discharge became foul in character. A third spinal tap was done, again negative for bacteria, and showed an increased cell count. The patient became irritable and decided she was throwing her money away on antibiotics, so during the month of February for the first time she was taken off antibiotics. On March 12, 1951, she was referred to us for treatment.

Examination at this time revealed a large postero-inferior perforation of the left drum. The canal was filled with yellowish-green foul, mucopurulent secretion. On mild suction after cleaning the canal

more mucopurulent secretion welled up through the perforation. The post-auricular scar was healed and tenderness of the mastoid tip and antrum were present. The spoken voice on the left was five feet. Rinne was negative and the Weber was to the diseased side. Stiffness of the neck and headaches were present, particularly when the drainage was lessened. All other findings were normal. Bacteriological studies revealed anhemolytic Streptococcus and Staphylococcus aureus, coagulase positive. The x-rays showed evidences of surgical removal of the cells posterior and inferior to the antrum. The Stenvers view gave the impression that the antrum had not been opened and large retro-sinus, dural, zygomatic and tip cells were present. Because of the comparative insensitivity of the organisms to all but massive doses of penicillin, the patient was placed on 1,200,000 units daily for one week. Because no real improvement either in symptoms or amount of discharge occurred, the patient was operated on for a third mastoidectomy in six months. On March 27, under endotracheal anesthesia, simple Lempert endaural incisions were performed creating a mobile extracartilaginous window. A 2.5 centimeter defect in the mastoid cortex was exposed posterior and inferior to the antral area. The remainder of the mastoid cortex was removed exposing granulations which were necrotic and did not bleed easily. Trabeculae were soft and in some areas were necrotic. On inspection it was found the antrum had a slit-like exposure along the dural plate. Superior to this slit the dural plate was absent and necrotic dural granulations were present in an area about 1.5 centimeters in diameter. The surrounding dural plate was removed until normal dura was encountered. The antrum was exposed widely and the attic and zygomatic cells cleaned out. The sino-dural angle, retro-sinus cells, tip cells and retro-facial cells were found diseased and untouched. These were excavated. In the area under the facial nerve and approaching the jugular bulb an abscessed area was encountered. The lateral sinus plate was found diseased here and reddish granulations were seen on the sinus wall in an area about 1 centimeter in diameter. The sinus was not explored. The wound was left unpacked for continuous drainage. Postoperatively penicillin, 1,200,000 units daily, was continued until patient was discharged on March 30, 1951. Postoperatively the patient made a rapid improvement. The hearing returned to normal, the perforation healed and to date no headaches or discharged have recurred.

Resume: This is a case of acute mastoiditis which developed under antibiotic therapy and went on to develop extradural abscess, perisinous granulations and subacute meningismus in spite of the antibiotics. In the first two mastoidectomies the antrum had not

been opened adequately and the mastoid pathology had not been thoroughly eradicated. When a complete mastoidectomy was performed the mastoid disease was healed and the subacute intracranial complications cured.

CASE 2.—*Otitis media, acute, meningismus, lateral sinus thrombosis, pachymeningitis externa both fossae, hydrocephalus and blindness.* H. J., aged 6, was referred from an outside clinic with the following history: Patient had been under treatment for two weeks and was given various sulfonamides and penicillin. He was lethargic and had tenderness of the right mastoid. The child was hospitalized immediately and a myringotomy was done. The penicillin was increased to 800,000 units daily and streptomycin, 250 mg every four hours, was added for four days. Aureomycin was given for 48 hours, a total of 1000 mg daily. Five days after hospitalization, on March 28, 1950, his temperature was normal and a definite fluctuation over the mastoid periosteum was found. A mastoidectomy was performed. The tip and the entire wall of the lateral sinus were broken down. A drain was inserted and the wound was closed. The wound healed nicely. Seven days after this surgery at the referring clinic he developed alternating periods of good vision and total blindness.

With this history the patient was referred to the Illinois Eye and Ear Infirmary on April 8, 1950. The patient appeared lethargic, his pupils were extremely dilated and he complained of loss of vision. The examination revealed a reddened and bulging right drum membrane and a linear scar behind the ear. He had a conductive type of deafness and an occasional spontaneous horizontal nystagmus to the right side. There was a severe nuchal rigidity, a postive Kernig and Brudzinski sign. The pupils did not react to light. He had a four-diopter papilledema with some superficial hemorrhages. The spinal fluid was clear and over 400 mm pressure. There were no cells in the spinal fluid. Pandy was negative. The total protein was 20 milligrams; no growth was present on culture. The blood count showed 4,600,000 red blood cells and 14,100 leukocytes. His temperature was 98.8°. His pulse rate was 72. Roentgen films of the mastoids showed a few periantral cells and a large area of destruction over the lateral sinus. The left side showed good pneumatization. Skull films suggested a questionable early destruction of cranial sutures as a result of increased intracranial pressure.

Under general anesthesia a T-shaped incision was made posteriorly. The cortex of the mastoid was rough and showed a fistula ½ centimeter in diameter which led to the anterior wall of the sinus, which was lying exposed about 1 centimeter below the upper knee.

The wall of the sinus was thick, necrotic and covered with granulations. No free pus was found. The bone anterior to the sinus was not removed by previous operation; it was necrotic and soft, partially destroying the anterior wall of the external canal. About one centimeter behind the antrum the tegmen showed a large fistula leading to the dura mater of the middle fossa. After removal of the tegmen this area of the dura appeared gray, necrotic and granulating. It was felt that this area was probably mistaken for the antrum at the first operation. The sigmoid sinus was then exposed for about 2.5 centimeters posterior to the knee until blue, healthy wall of the sinus was seen. Toward the jugular bulb the bone was very soft and was removed as far as it was possible to reach the posterior bony ring of the jugular bulb. The sinus wall itself was thick and covered with granulations. No normal wall was seen. The dura of the middle fossa was uncovered completely by removal of the tegmen of the mastoid, antrum and middle ear. The dura appeared necrotic and so was the area covering Trautman's triangle leading to the dura of the posterior fossa. Puncture of the temporal lobe revealed no pus. Puncture of the bulging and necrotic posterior fossa in the area corresponding to the lateral cysterna caused a free flow of cerebrospinal fluid. The lateral sinus was then aspirated. The transverse sinus contained blood about three centimeters posterior to the knee. No blood could be aspirated by puncture of the jugular bulb. After Whiting's tamponade was performed, the thickened wall of the sinus was slit and a red, friable thrombus was removed about 4 centimeters long. The wall of the sinus was partially excised and an iodoform gauze pack was inserted.

After surgery the patient became more alert, but the impaired vision and the bilateral papilledema with hemorrhagic exudates persisted. There was a beginning separation of the coronal sutures visible on x-ray. The patient was on penicillin therapy. His temperature ranged between 99° and 100,° and the pulse rate between 110 and 120. Because of persisting papilledema, blindness and intracranial pressure, which did not diminish after daily spinal punctures, the patient was transferred to the neurosurgical department at the suggestion of the neurologist for constant ventricular drainage.

Six days after the mastoid surgery the pressure of the cerebrospinal fluid was recorded at 480 millimeters. It was therefore felt by the neurosurgeons that an exploration of the right temporal lobe and a ventriculogram should be performed to rule out the possibility of a cerebral abscess. The procedures of puncturing of the brain and the ventriculogram were performed through a burr hole placed on the temporal squama and enlarged with rongeurs to about 3 cm

in diameter. The dura was found to be under pressure. It was then opened and the contents presented were normal in appeaarnce. No abscess was found.

After the surgery in the temporal squama was performed the incision was closed in layers. Two incisions were then made in the posterior parietal region about 3 cm in length. Here through burr holes the dura was opened and ventricular needles were inserted into the ventricular cavity. After 20 cc of fluid was removed from each lateral ventricule, air was inflated. The usual x-ray views revealed symmetrical ventricles of normal size and shape. There was a slight improvement of his eyesight. The patient was able to recognize light definitely and the papilledema showed definite signs of subsiding. At the time the spinal fluid pressure was recorded at 270 mm, the ventricular drainage was discontinued seven days after surgery.

Four weeks after surgery examination of the eyes showed that the right pupil reacted directly to light. Hand movements at six inches were recognized with difficulty. The left eye had no light perception. Fundoscopy revealed the venous engorgement markedly decreased. The retinal hemorrhages had been absorbed. Both nerve heads showed slight elevation and blurred margins. The discs appeared practically white.

The ear cavity granulated well and was healed completely ten weeks after admission. At this time the drum was retracted. The hearing showed an average loss of 40 decibels audiometrically in the speech range. Because of complete blindness the patient was transferred for his education to a school for the blind.

The patient was seen recently, one and one-half years after surgery. Bulging of the temporal area was present at the site where squama was opened for cerebral exploration. His ear was dry and his hearing was as it was postoperatively. The right eye still had light perception, but very poor projection. The left eye had no light perception. Examination of the fundus showed atrophic discs with white and ragged margins, demonstrating typical secondary optic atrophy.

Resume: This patient's acute otitis media was treated with penicillin and sulfonamides for two weeks. Under this therapy the patient developed mastoiditis. Then in spite of further extensive antibiotic therapy and surgery sinus thrombosis, hydrops and permanent blindness resulted.

CASE 3.—*Otitis media, acute, subtentorial purulent meningitis, facial paralysis, purulent lateral and cavernous sinus thrombosis.* M.

B., a 65-year old tabetic, white male was transferred to the Illinois Eye and Ear Infirmary from a mental institution with the following history: The patient became acutely ill two weeks prior to admission. He complained of headaches and vomiting. He ran a temperature varying between 100° and 102° and was placed on penicillin therapy for thirteen days. At this time he developed a purulent discharge from the right ear and progressively became apathetic, had mild stiffness of the neck and a slight facial lag on the right side. He was seen by an ear consultant who diagnosed mastoiditis and meningitis. The spinal fluid disclosed a 465 cell count and a very light growth of gram-positive pneumococci.

On admission to the Illinois Eye and Ear Infirmary it was found that the patient was disoriented. His temperature was 100.8° and his pulse rate was 74. There was rigidity of the neck and a Babinski sign on the right. The right ear was filled with purulent secretion. The canal appeared narrowed from a sagging of the posterior canal wall. Hearing tests could not be performed. There was no spontaneous nystagmus. Cold water calorization showed vestibular function had been preserved.

The left ear canal and drum appeared normal. Fundoscopy revealed no pathology of the discs or vessels. Spinal tap revealed 270 cells with 226 mg per cent protein. The blood count showed 11,350 white blood cells, 91.2 per cent hemoglobin and 4,330,000 red blood cells. Roentgen film in Schueller's position showed a large defect in the sinus plate with erosion posteriorly and anteriorly to the sinus. The antrum appeared enlarged and the remaining cells were small and hazy.

Surgery of the ear was performed postauricularly under local anesthesia. The antrum and the periantral cells were filled with pale granulations. The diploetic bone was also diseased. After removal of the lateral attic wall, the tegmen of the middle ear and the antrum appeared soft and necrotic. The overlying dura appeared covered with granulation (Pachymeningitis externa).

Similar dural changes of the posterior fossa were exposed after removal of the bone lateral to Trautman's triangle. A perisinous abscess covered the sigmoid sinus wall. Puncture revealed a thrombus which obstructed the middle and lower thirds of the sinus. Full bleeding was encountered at the sinu-dural angle and from a large emissory vein. No bleeding was noted from the bulbar end of the sinus. The thrombus and the granulating wall were excised.

Immediately postoperatively the patient showed some improvement. He was more alert and became afebrile on the second day after

surgery. On the sixth postoperative day he became restless and stuporous. He developed difficulties in urination and the meningeal signs, such as neck rigidity and a left Babinski. A spontaneous horizontal nystagmus of the first degree to the operated side, changing from time to time to the left, was noted. At the suggestion of the consultant neurologist, puncture of both fossae were performed. No pus was found in the cerebrum.

On the 17th day after surgery the patient was semicomatose and developed a great deal of mucopurulent secretion in the bronchi. Suction of the secretion could not be properly applied and a tracheotomy was done to relieve the increased restlessness and signs of anoxia.

The patient became progressively worse and expired on the 20th hospital day.

At necropsy a basal purulent meningitis was found extending from subtentorial meninges secondary to the right mastoiditis. Purulent infection of the right cavernosus sinus secondary to the sinus thrombosis was present. The cause of death was related to the purulent bronchitis and pulmonary edema superimposed on the meningitis, and extensive intracranial venous thrombosis.

Resume: This patient was a tabetic and probably had a silent complication from the ear at the onset. He was given penicillin for 13 days and the symptoms of the complication were further suppressed until obvious meningeal symptoms developed and the discharging ear disclosed its connection with the intracranial infection.

HISTOPATHOLOGY

In reviewing sections from bone of the mastoid and from the sinus and tegmen plate of these cases, it was found that new bone formation generally prevailed and osteoclasis was absent. Some cell lumina appeared obliterated, others filled with scanty exudate. The mucosa appeared thick and edematous and submucosa in places fibrous. Lymphocytic infiltration prevailed. This picture of bone pathology is usually seen in repair except for the complete lack of osteoclasis.

In addition to the osteal changes, vascular changes are noticed. Thrombi are seen in the precapillaries of the tegmen. This is in accordance with the findings of Macht and Pelz that penicillin favors thrombus formation. These facts raise the question whether these changes in the bone and vessels are not a sequence of antibiotics. We intend further studies in this direction.

CONCLUSIONS

In the management of acute otitis media penicillin has proven to be the drug of choice. This is in accord with the bacteriology usually present in these infections. In those cases in which penicillin is not immediately effective there is a tendency to haphazardly prescribe an array of antibiotics. This procedure of administering multiple antibiotics at the same time and without bacteriological and sensitivity studies has proven to be of little value and complicates the clinical picture. One should be especially alert at this point for the existence of unsuspected complications.

These few selected cases presented from among the many problems of otogenic complications seen by us at the Illinois Eye and Ear Infirmary during the era of antibiotic therapy serve to dissipate the illusion that mastoid disease is always prevented or cured by antibiotics. They also serve to illustrate the need for adequate surgery when surgery of the mastoid is indicated.

As temporal bone surgery has been perfected from Wilde's incision, through Schwartze's antrotomy to modern day techniques, so have the percentages of results of surgery improved. This principle has been lost sight of to the degree that undue reliance is placed on antibiotics to complete the treatment. It has been our experience that when surgery becomes indicated antibiotics have failed to prevent complications or effect a cure. Once this condition obtains, the surgery performed should be as thorough, meticulous and complete as the standards of pre-antibiotic days indicated.

V

ANGIOMAS OF INTEREST TO THE OTOLARYNGOLOGIST

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Angiomas may be classified in two large groups: (1) hemangiomas, or tumors of blood vessels and (2) lymphangiomas. The former are encountered far more frequently. Hemangiomatous elements are often seen in association with lymphangiomas; occasionally the reverse is true. The majority of angiomas occur about the head and neck.[1, 2] Most of these involve only the skin and subcutaneous structures but some involve both skin and mucous membrane. Some, particularly the lymphangiomas, are located solely within the mouth and upper part of the respiratory tract.

ETIOLOGY

Normal blood vessels and the earliest blood cells arise from the mesenchyme. The isolated masses and cords, or blood islands, from which this formation takes place collectively make up the angioblast. These angioblastic masses and cords undergo canalization; the cells in the center become the early blood corpuscles, while those at the periphery remain as flat endothelium. These new vascular spaces grow and unite with others, forming plexuses. Eventually a system of closed vessels with blood circulating through them is evolved and subsequently new vessels are formed only as outgrowths of pre-existing channels. Not all of the original networks and plexuses remain; many undergo atrophy, while those that remain become dilated.[3]

It has been suggested[2, 4] that hemangiomas may develop as embryonic sequestrations of portions of the angioblast, that is, failure of a group of these angioblastic cells to make contact with the other

Read at the meeting of the American College of Surgeons, New York, New York, September 22 to 26, 1952.

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groups that are forming networks. These isolated groups of cells form independent networks of blood or lymph vessels or both that are considered to grow by means of projecting buds of endothelium. Angiomas thus are new growths and should be distinguished from vascular enlargements such as chronic varicosities and aneurysms and also from such self-limiting processes as the formation of hypertrophic granulation tissue.[5] Angiomas are also to be differentiated from excessive development of blood vessels such as occurs in certain tumors as a result of venous stasis or rapid growth of the tumor.

Watson and McCarthy,[2] in a study of 1,056 cases of angiomas, found that evidence of the tumor was present at birth in 73 per cent and stated that hemangiomas make up the largest single group of neoplasms occurring in childhood. Pack and Miller[4] have estimated that three fourths of these tumors are present at birth and that in many other cases they appear soon after birth. At first the lesion may appear to enlarge rapidly but generally it enlarges only in proportion to the growth of the child. Great enlargement of these tumors may be observed at the onset of pregnancy or less frequently at the onset of menstruation.

Most observers[1, 2, 4, 6] have found that two thirds of hemangiomas occur in females. This has prompted speculation as to an etiologic relationship to the female hormones. Study of a large number of cases revealed that multiple lesions were present in 16 per cent. Hemangiomas are seen rarely in negroes.

CLASSIFICATION AND PATHOLOGY

Hemangiomas are benign growths and may be divided into two main groups, capillary and cavernous.

The capillary hemangiomas include: (1) nevus flammeus or port-wine stain; (2) hemangioma simplex or strawberry birthmark; (3) nevus araneus (spider angioma or stellate hemangioma) and (4) senile angioma (deMorgan's spots).

Port-wine stains are flat, pink to bright-red, capillary hemangiomas, usually seen about the face, especially the upper lip. They may involve also the oral mucous membrane. In one large group[2] of cases, 70 per cent of these lesions were in males. Enlargement of the involved region usually is caused by an associated underlying cavernous hemangioma. Microscopic examination of the capillary hemangioma reveals a narrow zone of dilated vessels lined with endothelium of adult type. A port-wine stain is sometimes observed in association with signs of cerebral cortical dysfunction and typical

roentgenologic findings in the skull. This combination constitutes the Sturge-Weber-Dimitri syndrome.[7, 8]

Hemangioma simplex is a slightly raised, circumscribed, finely lobulated, bright-red tumor. The solitary small strawberry birthmark is the classic example of it. This tumor often accompanies the infantile form of laryngeal hemangioma.[9, 11] It is composed of dilated vessels of the capillary type, that is, a single layer of endothelial cells forms the walls of the vessels.

Spider angiomas are small, capillary lesions that have a central vessel of varying size (up to 2 or 3 mm in diameter) which may be elevated above the surrounding surface. From this central hub fine hairlike vessels radiate. These lesions have a fiery-red color because they arise from arteries in the subcutis. They are of interest because they are indistinguishable from the telangiectatic regions seen in hereditary hemorrhagic telangiectasia.[12, 13]

Cavernous hemangiomas are soft compressible tumors. They are composed of greatly dilated blood vessels with pools and sinuses; it is generally considered that there is only one afferent and one efferent connection with the general circulation.[4] They may be of great size depending on their site and vascular connections. The vascular defect can occur at the time a limb bud is forming so that all structures of the limb including muscle and bone are involved by the hemangioma. This has been termed "hemangioma unius lateralis." Similarly a hemangioma also may follow the cutaneous pattern of a peripheral nerve, as the trigeminal.

Racemose hemangioma, also called "plexiform hemangioma" and "cirsoid aneurysm," is a special form of hemangioma that usually has a connection with the carotid artery. It has been described as being like a pulsating mass of earthworms. Often it extends over the neck and scalp and by the pressure of its pulsations it has been known to erode bone and to establish connection with the meningeal vessels.

Lymphangiomas may be classified in the same manner as are the hemangiomas. First, corresponding to the simple or capillary hemangiomas, there is the rare circumscribed warty growth in the skin called "simple lymphangioma." Secondly, there are the cavernous lymphangiomas. Most lymphangiomas are included in this latter group. They are composed of dilated lymphatic sinuses that have a flat endothelial lining. The amount of stroma varies; some lymphoid tissue and occasionally a small amount of smooth muscle may be present. Like the cavernous hemangiomas, these tumors may involve

the mucous membranes of the oral cavity, and also the lip, tongue and cheek. Lymphangiomas may involve an entire extremity just as their hemangiomatous counterparts do.

A final type, considered by many to be a cystic form of lymphangioma, is cystic hygroma.[2, 5] These tumors are compressible masses of thin-walled, multilocular cysts that are filled with serous fluid and lined by endothelium. They usually begin in the submaxillary region, then ramify up to the parotid gland and mastoid region, medially to the midline and down to the clavicle and even into the interior of the thoracic cage.

CLINICAL ASPECTS

In general, the symptoms caused by angiomas are owing to their site and size, whether or not they may be easily traumatized, how much they interfere with the normal function of the part involved and, lastly, to their appearance. Serious hemorrhage may occur as a result of trauma. Recurring acute infections in angiomas are not uncommon.

In this presentation, any special comment will be limited to those lesions which occur in the field of the otolaryngologist.

Ear.—Reports of hemangiomas affecting any part of the ear are infrequent. Occasionally a cavernous hemangioma is seen that encompasses the entire auricle. Weaver[14] reported such a case in 1943, and, in 1947, Pirodda[15] reported a racemose arterial hemangioma of the auricle. These tumors frequently become ulcerated and infected. Severe hemorrhage may occur, requiring ligation of the carotid artery and at times removal of the whole auricle.

Such a case is under our observation at present. Previous attempts to isolate and ligate all afferent vessels had failed, as had also ligation of the external carotid artery. The use of sclerosing solutions had been without benefit. Prolonged infection had resulted in perichondritis. Hemorrhage had occurred frequently and at times had been severe. We removed almost the entire auricle after having made total ligation at its base. The stump of the amputated auricle has healed and at this moment the problem seems to be under control. It is too early however for a final report because in this case the possibility exists that an arteriovenous fistula may be present.

Hemangiomas may occur in the middle ear and mastoid[14] but are rare in this location.

Nose and Sinuses.—The septum rarely is the site of a cavernous hemangioma. It may be implicated by a portion of a larger tumor

of the external part of the nose, as in Salinger's[16] case, or the tumor may be a smaller one confined to the septum. Sooy[17] recorded a case of the latter type of tumor and stated that such lesions usually are polypoid in appearance and are located in Kiesselbach's area. Réthi[18] stated that he had seen five cases of bleeding septal polyps that had the appearance of a mushroom and, like the hemangioma in Sooy's case, were found in Kiesselbach's area. These probably were hemangiomas.

Bleeding polyps of the septum were described by Thomson and Negus[19] as granulomas of doubtful etiology that have undergone angiomatous change. From their description it is not possible to determine whether they had observed true angiomas or pseudo-angiomas (excess granulation tissue).

The nasal septum frequently is the site of the spider angiomas of hereditary hemorrhagic telangiectasia. This familial disease, which is also called "Rendu-Osler-Weber disease," is thought by many to be a congenital anomaly involving the walls of small blood vessels in localized regions. The disease is transmitted by either sex as a simple dominant and may skip generations or individuals. Pack and Miller[4] listed the following triad as essential for the diagnosis of this disease: (1) mucosal hemorrhages (usually epistaxis), (2) familial occurrence and (3) small telangiectatic angiomas of the skin and mucous membranes, usually those of the oral and nasal cavities. It has been suggested that a fourth requirement be added, which is the presence of a normal blood picture (except changes owing to loss of blood, if it has occurred) and normal bleeding and clotting times.

The cutaneous lesions of hereditary hemorrhagic telangiectasia are described as vascular areas that are punctate, round, macular, sessile and not usually raised, sometimes spider-like and not larger than 3 mm in diameter.[4] Such lesions are similar to the telangiectatic spots commonly seen in the skin of normal elderly people. The common sites of the cutaneous lesions are: cheek, nasal orifices, ears, finger tips and beneath the nails. At first the areas are bright red; later, they tend to become purple.[13] Lesions of the mucous membrane are always bright red. Often the lesions are flat but many of them have a slightly raised center; they are found most frequently on the cartilaginous portion of the nasal septum, usually on both sides.[12] They may be observed fairly frequently on the tongue (dorsum and tip), lips, palate, pharynx and buccal surfaces, less frequently on the gums and conjunctiva, and occasionally in the larynx, stomach, colon and bladder.[20]

In discussing this disease most authors[4, 6, 12, 20] agree that, although the history of epistaxis usually dates from puberty, the onset of severe hemorrhages and the presence of clinically evident telangiectasia are noted most frequently in persons of the fourth decade of life or later. The capillaries of the telangiectatic lesions are dilated and thin walled and profuse hemorrhage can occur from the nose spontaneously or after such slight trauma as a sneeze or a cough. The epistaxis may be a steady ooze or profuse hemorrhages may occur at varying intervals of hours or days. The secondary anemia thus produced increases slightly the tendency to bleed.

Although hemorrhage in this disease usually is from the nose, similar persistent or repeated bleeding from the lips and from the gums has been reported.[4, 20]

Réthi,[18] in commenting on hemangiomas of the upper part of the respiratory tract, reported a case of "cavernoma" of the inferior turbinate and two cases of "cavernoma" of the maxillary sinus, in one of which the diagnosis was confirmed by microscopic examination. Weaver,[14] in his report on three cases of unusual hemangiomas, described a capillary hemangioma in the region of the ethmoid sinus.

It has been noted that hemangiomas of bone are rare. Most of them are asymptomatic because they are small and grow slowly. The bones of the skull are said to be attacked more frequently than other bones[4] and several such lesions have been reported. Neivert and Bilchick[21] reported what they considered to be the only recorded case of primary hemangioma of a nasal bone. Their patient had pain and swelling over the side of the nose and roentgenologic examination of the nasal bones revealed numerous fine reticulated lines. The tumor was removed surgically with complete cure and no deformity.

In 1947, Erich[22] reported one case of central hemangioma of the mandible. He pointed out that such a lesion is extremely rare and that the diagnosis can be made only by biopsy.

Mouth and Pharynx.—It is unusual to see a hemangioma that invades only the oral cavity or pharynx, but it is common for such lesions to be present in conjunction with a hemangioma of the face or neck. These lesions usually are of the cavernous variety, but a port-wine stain of the face may extend into the mouth. We never have seen a hemangioma simplex inside the mouth but undoubtedly such lesions do occur. Frequently a cavernous angioma will underlie a port-wine stain and both may include the lip and buccal mucosa.

Fatal hemorrhage has been reported after dental extractions in patients in whom the mouth was attacked by a cavernous hemangioma.[23, 24]

Hallberg,[25] in a study of 409 solid tumors of the palate, found 11 angiomas. Eight of these were hemangiomas and the other 3 lymphangiomas. Of the 8 hemangiomas, 4 were isolated lesions confined to the palate. Five of the 8 patients who had hemangiomas received treatment with radium and a good result was reported in each instance.

Smith,[26] in 1948, reported a case of cavernous hemangiomas of the soft palate. Treatment by injection of a sclerosing solution gave excellent results.

Cavernous hemangiomas of the tongue are fairly common and the tip of the tongue has been said to be the portion most frequently affected. Such a tumor can become greatly swollen during episodes of acute inflammation and thus seriously interfere with function, or even cause respiratory obstruction.

New and Childrey[27] made a detailed survey of 357 tumors of the tonsil and pharynx. In this series, two hemangiomas and one lymphangioma were found. The two hemangiomas were classified as fibroangiomas on microscopic examination but it was stated that the fibrous element did not predominate in either. Both tumors were found in children, a girl seven years of age and a boy 10. One tumor was situated high on the posterior pharyngeal wall and caused nasal obstruction, while the other was situated low and caused laryngeal obstruction. Both were purplish blue, whereas the lymphangioma was pale pink.

Lillie and Heck[28] reported a case of hemangioma of the lateral pharyngeal band. The chief complaint was cough, which was worse at night. The authors suggested that the cough was caused by vagal irritation and postulated that the nocturnal increase in severity was produced by engorgement on lying down.

Walford[29] reported the case of a patient whose presenting complaint was mild hemoptysis. A small hemangioma was found on the posterior pharyngeal wall. The tumor was removed by means of surgical diathermy.

The lips, cheek and tongue are frequent sites of cavernous lymphangiomas. These tumors can therefore cause macrocheilia, macromala or macroglossia. They produce irregular and sometimes extensive enlargements and have a finely nodular appearance, some-

what as if numerous small kernels of rice lay immediately beneath the mucous membrane.

Nasopharynx.—Juvenile fibroma is mentioned because it is a highly vascular lesion and because some evidence suggests that it may originate in or from a pre-existing angioma. Nasal obstruction and occasionally severe epistaxis are the two major symptoms of juvenile fibromas of the nasopharynx. These tumors occur almost exclusively in males;[30, 31] some authors[32] say they occur only in males. The lesions appear at or about the age of puberty and tend to regress spontaneously when sexual maturity is reached. According to Martin and associates,[32] the angiomatous element of these tumors predominates in younger subjects and these authors postulated that the lesion probably begins as an angioma rather than as a fibroma. They were of the opinion that at first the fibrous portion is the usual stroma for the abnormal blood vessels but that the fibrous component later increases with changes in the sex hormones. This apparently occurs also in the so-called dermatofibromas or sclerosing hemangiomas. This opinion as to the histogenesis of these tumors is reflected in the name used by Martin and his associates, "Juvenile nasopharyngeal angiofibromas." The use of terms such as "vascular fibroma"[33] and "bleeding fibroma of adolescence"[32] to designate this lesion indicates that other observers have the same concept as to its nature.

Réthi[18] described an interesting hemangioma of the epipharynx. This tumor occurred in a boy, 17 years of age. It was said to arise from the roof of the epipharynx and could be seen as a plum-sized, greyish-red tumor that had a smooth surface and was surrounded by granulation tissue. The microscopic diagnosis was cavernous hemangioma. This case would appear to be similar to one of the previously mentioned cases of New and Childrey.[27] In the latter case, the lesion was high on the posterior wall and produced nasal obstruction. Their patient had not yet reached the age of puberty. Perhaps a small symptomless similar lesion might enlarge at puberty and become the lesion recognized as juvenile fibroma.

Larynx.—Hemangiomas of the larynx have been classified[34] as infantile or adult. The infantile type is found in the subglottic region and the prominent symptom is dyspnea; in the adult, the tumors are almost always above the vocal cords and the main symptom is hoarseness. Most of these hemangiomas are cavernous in nature. More often than not they are associated with similar conditions in other regions of the upper part of the air passages.[10]

In reviewing the literature up to 1944, Ferguson[9] found that 8 cases of tumors of the infantile type had been reported and that in three of these hemangiomas were noted elsewhere in the body. He also found reports of 123 cases of hemangioma of the larynx. New and Erich[11] found 24 hemangiomas in 722 cases of benign tumor of the larynx. Two were infantile and 22 were adult in type. More recently, Holinger and Johnston[35] studied 1,197 benign tumors of the larynx and found four to be hemangiomas. In 1949, Berntsen[36] described two cases, in one of which the lesion was a large cavernous hemangioma in a 71-year-old woman who had been hoarse for sixty-five years.

Sweetser,[34] who reviewed 38 cases of laryngeal hemangioma in 1921, found that 66 per cent were in men. Sixty-eight per cent of the 24 cases reported by New and Erich[11] were in males. It will be remembered that, as stated earlier, two thirds of all hemangiomas are in females. This reversal of sex incidence was noted also in port-wine stains and angiofibromas of the nasopharynx.

Hemangiomas of the larynx in the adult usually are small, nodular, polypoid and often pedunculated. Their color is bluish, reddish or pale pink, and the average diameter is 5 to 6 mm. The infantile growths are flattened, sessile, diffuse and dark blue or purple.

Hoarseness, cough, dyspnea, hemoptysis and dysphagia in that order are the common symptoms, according to Ferguson.[9] New and Erich[11] stated that the only symptom of any diagnostic significance was hemoptysis.

Lymphangiomas are much less common in the larynx than are hemangiomas. Cordray and Gervais,[37] in 1951, found only 6 cases and added one. Holinger and Johnston,[35] in 1951, and Bernhardt,[38] in 1949, each reported one case of lymphangioma.

New and Erich[11] noted only one case in their large series. Most of the lymphangiomas reported have been described as larger than the average hemangioma. They are also smoother and are pale in color or transparent in appearance.

Tracheobronchial Tree.—Hemangioma of the tracheobronchial tree was merely listed as one of the possible benign tumors by Thomson and Negus.[19] Jackson and Jackson[39] expressed the opinion that many of the tumors previously reported under such designations as hemangiomas and fibromas were really adenomas of the bronchus.

Esophagus.—It is well known that any benign tumor in the esophagus is a rarity. Patterson,[40] in an exhaustive review of the literature in 1932, could find only 61 cases and in this group only one hemangioma was noted. Moersch and Harrington,[41] in 1944, included mention of three hemangiomas in their report on benign tumors of the esophagus. Ferguson and Hackworth[42] recorded a case of hemangioma of the esophagus in a child.

Sturge-Weber-Dimitri disease is of interest to the otolaryngologist because the superficial hemangiomatous lesions that form a part of this disease or syndrome may occur in the mucous membranes of the mouth, nose, throat or conjunctiva, although usually this unit of the triad of findings is a port-wine stain of the face and scalp. The two additional findings necessary for a diagnosis of this disease are signs of cerebral cortical dysfunction and the presence of sinuous shadows in roentgenograms of the skull. McMahon,[8] in 1951, reported a case in which an infant had lesions affecting one side of the forehead, external ear, mouth and pharynx. Stridor was present and subglottic stenosis was noted on the roentgenogram. This disease has also been called "encephalo-trigeminal angiomatosis."[7] Apparently a focus of hemangiomatous dysplasia is present in the layer of mesenchyme directly bordering the ectoderm that forms the skin of the cephalic region, the mucous membranes of the mouth, nose and throat, the choroid coat of the eye and the leptomeninges enveloping the brain. This disease is often grouped with two other neurocutaneous syndromes, namely, von Recklinghausen's disease and tuberous sclerosis.

TREATMENT

As has been stated by Figi,[43] treatment of an angioma may be indicated for the following reasons: (1) cosmetic purposes, (2) rapid extension of the lesion, (3) interference of the lesion with function, (4) repeated spontaneous or traumatic ulceration, (5) danger of serious hemorrhage or infection and (6) possibility of malignant change.

Two general plans of treatment are available. The first of these is complete removal of the lesion by excision or by electrocoagulation. Excision may be followed by either primary suture of the wound or application of a skin graft. Removal by electrocoagulation is applicable only for small lesions.

The second general plan is designed to obliterate the lesion by use of some agent to induce fibrosis. Several means and agents are available for this purpose. The most important are: (1) injections

of sclerosing solutions or of boiling water; (2) ligation, either alone or in combination with injections; (3) deep electrocoagulation, which is not to be confused with the destruction of an entire small lesion just mentioned; (4) irradiation with roentgen rays or with radium applied to the surface or interstitially and (5) refrigeration by application of carbon dioxide snow.

Surgical excision may be employed for those lesions which are well localized, regardless of the fact that they may be extensive. It is seldom that any but capillary angiomas or small cavernous angiomas are suitable for this form of treatment.

Injection of sclerosing solutions,, among which is a 5 per cent solution of sodium morrhuate, is suitable only for cavernous angiomas and is particularly suitable for those lesions in which unusually large vessels are present that make pools of blood and in which suitable compression can be used to stop temporarily the circulation of blood through these pools while the injection is being made. Boiling water is not often found to be a desirable agent for injection.

Ligation may be employed in either of two ways. In one, an attempt is made to identify and to ligate the afferent vessel. If this can be accomplished, subsequent pressure over the lesion to keep it collapsed will result in an obliterating fibrosis. Injection may be combined with ligation. Identification of the proper vessel is difficult, however, and this method is not especially satisfactory.

The second method for ligation consists of the use of multiple ligatures placed throughout the lesion by means of a ligature needle. A deep "bite" is taken and then the needle is made to re-enter the skin through the wound of exit and pass superficially to emerge through the wound of entrance. The ligature is then tied and the knot slips through the puncture wound to lie subcutaneously. Fine chromic catgut is used and an attempt is made to place a number of ligatures sufficient to include the entire lesion.

Deep electrocoagulation can be used to induce fibrosis by means of heat. A special electrode that consists of stiff wire with a sharp point must be used. The wire is covered with insulating material except at the point. This insulated needle is inserted into the depths of the angioma before the electrocoagulating current is turned on. When the current is applied, the tissues at the point of the needle are coagulated. The needle then is moved to another part of the lesion and the process repeated as often as necessary to provide coagulated portions throughout the angioma. Healing leads to fibrosis

and obliteration of the coagulated portions. If the angioma is large, it may be necessary to repeat the process at intervals of two to three months.

Irradiation by means of roentgen rays or surface application of radium may be employed. It is often of value in treatment of small superficial lesions but rarely is of much benefit for hemangiomas of the cavernous type.[44] Use of interstitial irradiation is valuable in treatment of this latter type of lesion. Radon seeds that have a value of 0.1 to 0.4 millicurie each are implanted throughout the lesion so that one such radon seed is present in each cubic centimeter of the tumor. If the radon seeds can be thus accurately placed, the resulting fibrosis will provide a most gratifying result in a great number of cases. The fear has been expressed that use of irradiation for angiomas may in later life lead to malignant change in the irradiated tissue. We are of the opinion that this possibility is remote or nonexistent in connection with the use of interstitial irradiation as described.

Irradiation by means of roentgen rays or surface application of radium is also valuable in the treatment of lymphangiomas; except for situations in which excision or partial excision may be necessary, such irradiation is the treatment of choice.

Cystic hygroma is not a well-demarcated lesion; therefore, because complete excision may not be possible, treatment should first be by means of external irradiation. Often this alone will be sufficient but if not, subsequent excision will be aided rather than hindered by the fibrosis produced by the irradiation.

Treatment of angiomas by use of carbon dioxide snow is accomplished by applying a suitable-sized block of dry ice to the lesion with firm pressure for several seconds at intervals of a week to ten days. Repeated treatments are necessary until the desired result is obtained.

Treatment of Specific Lesions.—Treatment of port-wine stains by any means other than excision, which usually is followed by grafting of skin, is likely to prove disappointing. Excision and grafting of skin must be considered with caution because the grafted skin may be much whiter than the adjacent normal skin and thus be just as conspicuous as the lesion for which it was substituted. This is especially true in the case of pale-pink lesions; often it is better to leave these untreated and to instruct the patient in the use of cosmetics to conceal them.

Hemangioma simplex often can be excised. Surface irradiation often gives good results, as does also interstitial irradiation. The

response to irradiation is better in the early months of life than it is if the child is a year or more old.

Spider angiomas can be treated satisfactorily by electrocoagulation of the central vessel.

Cavernous hemangiomas may require various types or combinations of types of treatment depending on their size and location; ligation or injection or both, deep electrocoagulation and interstitial irradiation are the methods most likely to be of value.

The racemose hemangioma is a challenging lesion and attempts at treatment may prove discouraging. The mainstay for treatment of these lesions is ligation.

Angiomas of the external portion of the nose are best treated by interstitial irradiation. For lesions that involve the septum, it may be possible to employ interstitial irradiation or electrocoagulation. In the treatment of lesions that invade the nasal accessory sinuses, destruction by electrocoagulation and, if possible, use of interstitial irradiation should prove most satisfactory.

The lesions of hereditary hemorrhagic telangiectasia are treated by electrocoagulation of the central vessel of each spider nevus. The use of moccasin venom has been found to be of value in decreasing the tendency for the lesions to bleed.

The juvenile nasopharyngeal fibroma is a highly vascular lesion and its treatment must be approached with caution. Ligation of one or both external carotid arteries is advisable prior to any type of treatment. A highly satisfactory method of treatment, in the experience of one of us (Havens), is exposure of the tumor by an approach through the nasal passages or nasopharynx or both, electrocoagulation of as much of the tumor as possible and implantation of radon seeds. A transantral approach for exposure of the tumor may be employed and is preferred by some surgeons.

Hemangiomas of the larynx, since they tend to be small and often pedunculated, usually are suitable for treatment by removal and electrocoagulation or vice versa.

SUMMARY

The essential and commonly accepted opinions regarding angiomas have been given in a condensed form. Methods of treatment in general have been given, and for specific lesions in the field of otolaryngology we have indicated those methods of treatment which in our hands have been most valuable.

MAYO CLINIC.

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VI

A CORRELATED STUDY OF THE ANATOMY AND MOTOR ACTIVITY OF THE UPPER PHARYNX BY CADAVER DISSECTION AND BY CINEMATIC STUDY OF PATIENTS AFTER MAXILLO-FACIAL SURGERY

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Interest in the motor function of the pharynx has recently been stimulated by the increasing incidence of bulbar-pharyngeal poliomyelitis. The accumulation of patients having residual difficulties of deglutition and speech, sequelae of this form of poliomyelitis,[1] impels us to study further the motor mechanisms of the pharynx and to seek new methods of correction of these particular handicaps.

Extensive descriptions of the anatomy and function of the pharynx are available. These are based for the most part on dissections of the cadaver[2, 3, 4] or visual observations of the pharynx exposed by anomaly[4, 5] or by surgery.[6, 7, 8, 9] However, study of the pharynx is difficult, for it is a highly mobile organ whose action can be but grossly inferred from cadaver dissection and, in the living, the rapidity and complexity of its motion makes visual evaluation difficult. These studies correlate cadaver dissections and cinematic observation of activity of the pharynx exposed by anomaly or by surgery. They have also been undertaken in order to identify the motor phenomena which may be involved in poliomyelitis paralysis.

MATERIAL AND METHODS

The cadaver material under study had been prepared in the usual manner with injection of contrast media into the arterial system. All anatomical material upon which comment is made was based upon three or more dissections. Critical or minute sections were completed under magnification. The studies to be described were based upon dissections of the musculature of the upper pharynx and the eustachian tube in 18 cadaver specimens.

The cinematographic studies with which these anatomical observations were correllated were performed on 7 subjects. In six

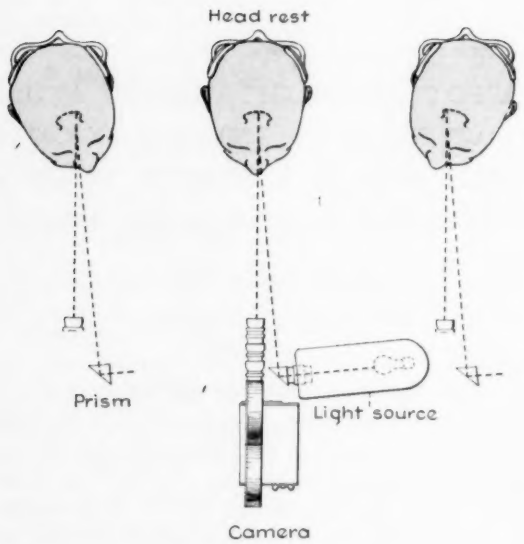

Fig. 1.—Diagram of relation of camera and light source as these were employed in the cinema study of the pharynx.

of these the upper pharynx had previously been exposed by surgical resection of neoplastic or osteomyelitic tissue in the area of the nose and paranasal sinuses. One subject had a wide unrepaired cleft of the hard and soft palate. Cinematic photography in these recessed areas was accomplished by means of a 16 mm camera with a 25 mm lens, at a focal distance of 18 or 22 inches. Light was provided by an air-cooled 1000-watt projector lamp with the central portion of the light beam angled by a vertical prism so that it nearly approximated the line of the camera* (Fig. 1). The subjects were partially immobilized in a dental chair. Standard patient-camera positions employed included the median or sagittal position for study of posterior structures and left and right oblique positions, to visualize the lateral areas of the pharynx. Visualization of lateral areas was facilitated by gentle manipulation, without anesthesia. The potential variety and range of motion of the palate was demonstrated by observation during phonation of a small variety of practiced sounds and during swallowing in those subjects in whom this area was surgically exposed.

*This apparatus was designed and constructed by Drs. William Kubeck and Frederick Kottke of the Department of Physical Medicine, University of Minnesota.

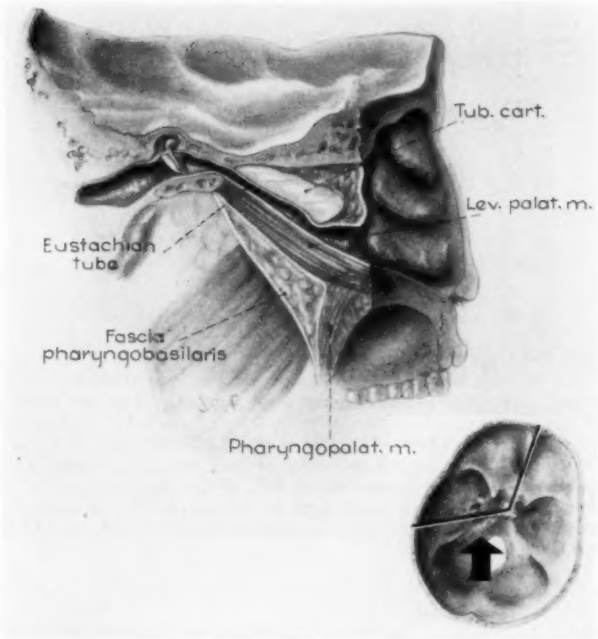

Fig. 2.—Artist's drawing of dissection of cadaver specimen showing position and relations of levator palatini muscle. Note this muscle lies at an oblique angle to the sagittal plane (insert diagram), so that its direction of pull of the palate is posteriorward, as well as upward and lateralward. The close relationship of the muscle to the eustachian tube is shown.

This report is concerned principally with the nasal and oral pharynx, by reason of the opportunity of surgical exposure of these particular areas and of the limitation of the comparison study of patients having poliomyelitis paralysis of the pharynx. In the interest of clarity, the complex musculature of this portion of the pharynx is classified somewhat empirically under two major categories—the *levator* and the *constrictor* groups. In as much as it was impossible to distinguish the function of the tensor veli palatini in the patients having surgical exposure or paralysis of this area this particular muscle is mentioned only incidentally.

THE LEVATOR MUSCULATURE

Anatomical Studies. The intrinsic group of *levator muscles* of the pharynx includes the levator veli palatini and the salpingo-

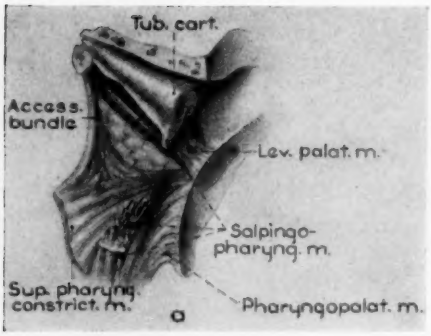

Fig. 3.—Artist's drawing of section of cadaver specimen showing position and relations of the tubal cartilage and the levator palatini, superior pharyngeal constrictor and palatopharyngeal muscles. Note the close relation of the levator muscle to the posterior wing of the tubal cartilage, from the inferior edge of which is suspended the salpingopharyngeal fascia and muscle (shown in cut section at border of cartilage and at interdigitation into musculature of the postero-lateral wall of the pharynx). The accessory bundle of the superior pharyngeal constrictor which originates at the base of the cranium with the levator is variant among the different dissected specimens.

pharyngeus muscles. The pharyngopalatine muscle, which has a vertical direction of action, is more closely related to the superior constrictor and will be considered in that association. The glossopalatine muscle appeared to contribute little to the functions considered in this study and was not separately dissected.

The *levator veli palatini* is one of the least variable muscles in the pharynx (Fig. 2 and 3). It arises from the base of the skull at the apex of the petrous portion of the temporal bone and extends in a single column into the palate, where it interdigitates with the insertions of the pharyngopalatine muscle, the tensor veli palatini, and the musculus uvulae. The levator muscles on either side are raised as a broad-based sling, which by reason of the relative posterolateral position of its origin (Fig. 2) acts to raise the central portion of the palate and to move it posteriorly.

The levator veli palatini muscle is related closely in position and function to the tubal cartilage, which is attached to the basal cranium on a line closely parallel to that of the levator. Within a deep groove of the anterior-inferior aspect of the cartilage lies the eustachian tube, which is also paralleled on its inferior aspect by the levator. The broad, flat tensor veli palatini covers much of the anterior aspect of the tube. The larger posterior wing of the car-

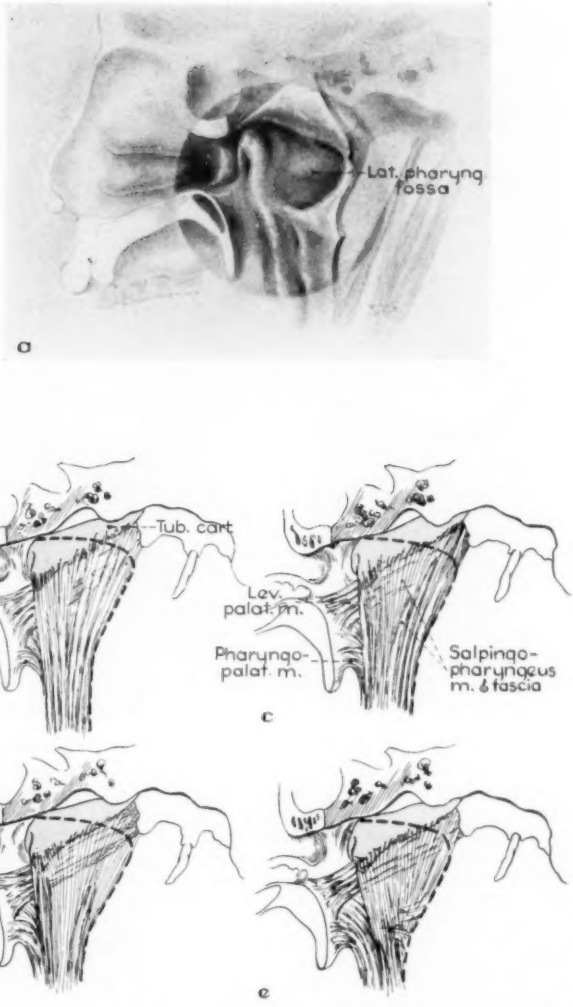

Fig. 4.—Artist's sketch of dissected cadaver specimens showing topography of lateral wall of the pharynx (a) and of variations in the muscle within the salpingopharyngeus fascia (b, c, d, e). The specimen in "a" shows with particular clarity the position of the pharynx in action. Note the relatively high position of the torus tubarius. The vertical ridge of the salpingopharyngeal fascia and muscle and the nearly horizontal ridge of the accentuation of the superior pharyngeal constrictor at the level of its sphincter contribution to the palatopharyngeal closure. Correspondingly, the palate would be in an elevated and posterior position, but has apparently been displaced downward and forward by postmortem manipulation.

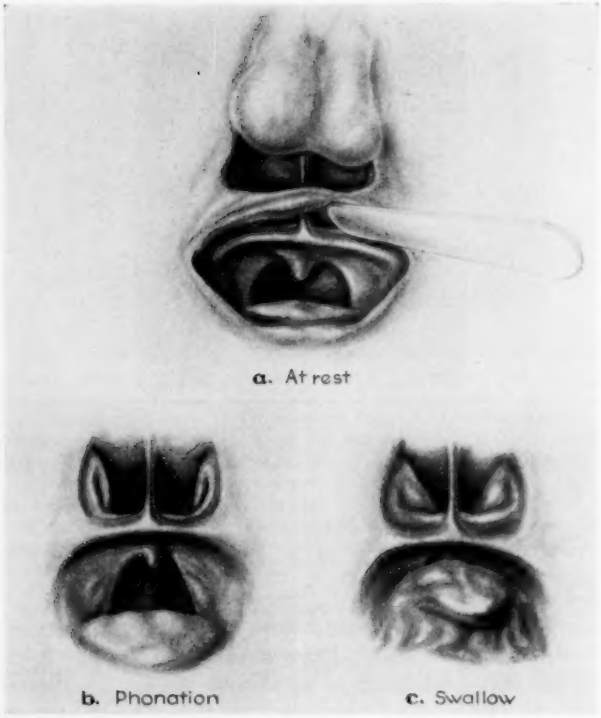

Fig. 5.—Pharynx of subject H. H. at rest (a) and position during action of phonation (b) and of swallow (c). Artist's drawings taken from selected frames of cinema film study. Subject, an adult female, underwent an extensive surgical excision of the nasal area for removal of a carcinoma, two and four years previously. The major portion of the hard palate, nasal septum and superior alveolar ridges were removed, leaving the posterior margins of the septum and hard palate. Currently, the patient wore a plastic prosthesis replacing the hard palate. With the aid of this appliance, she was able to swallow normally and had but a slight nasal quality of speech. Note convergence of torus tuberius coincident with elevation of palate and adduction of palatopharyngeal folds during phonation. During swallowing, further adduction of torus occurs and the mass of the levator muscle appears beneath it.

tilage, the torus tubarius, is roughly triangular in shape and overlies the posterior aspect of the levator. This posterior wing of the tubal cartilage gives origin to the salpingopharyngeal fascia and muscle which is suspended from the length of its inferior border. The muscle is inserted into the postero-lateral wall of the oral and laryngeal pharynx (Fig. 4). Despite careful dissection of these specimens,

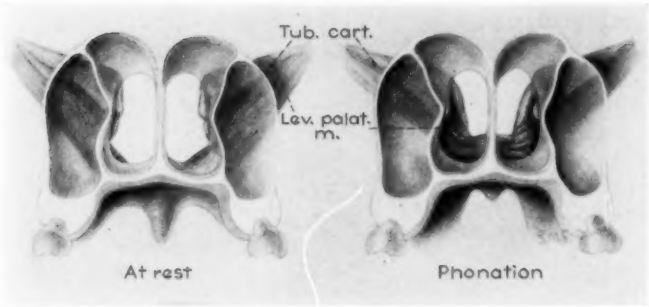

Fig. 6.—Artist's sketch of cadaver specimen which has transected at level of anterior margins of retained hard palate and nasal septum of subject H. H. (Fig. 5), and which was dissected to show relative position of tubal cartilage ("Tub. cart.") and levator palatini muscles ("Lev. palat. m."), which are shown in relief. In the right hand figure is shown the relative position of tubal cartilage, levator muscle and palate in simulated position of these structures in phonation.

no muscle could be found to correspond to the salpingo-palatine described by Simkins[10] as extending from the lower medial tip of the tubal cartilage to the adjacent palate.

As the contraction of the levator is simulated in the cadaver, it elevates the palate and also, through the tubal cartilage and the salpingopharyngeus fascia and muscle, elevates the pharyngeal tube as a whole, by reason of the angle of origin of the tubal cartilage and the shape of its posterior wing. This elevation of the cartilage is combined with adduction of the posterior wing of the cartilage and the salpingopharyngeus fascia and muscle suspended from it. The contribution of the salpingopharyngeus to the elevation of the pharyngeal tube is probably variable in different individuals, for the quantity of muscle within the salpingopharyngeal fascia in the nasopharynx is highly variable. This variation was compared in eight randomly selected cadavers. In two of these specimens (Fig. 4b) there was a prominent plane of muscle originating on the tubal cartilage and extending downward as a band in the pharynx, within the salpingopharyngeal fold. In four specimens there were two distinct bands (Fig. 4 c,d.), the one originating from the medial tip of the tubal cartilage and continuing downward in the medial edge of this plane, and the other originating on the spinous process and adjacent lateral portion of the tubal cartilage and extending downward and medialward in this fascial plane and in the lateral wall of the nasopharynx. In one specimen (Fig. 4e) the salpingo-

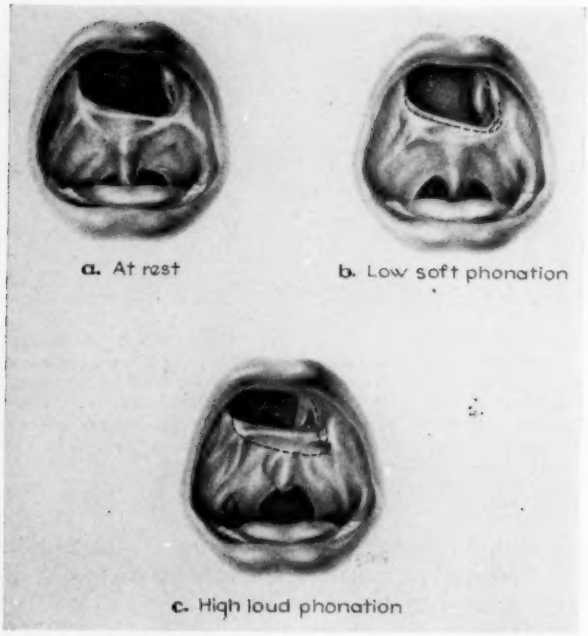

Fig. 7.—Pharynx of subject G. R. at rest (a) and in position of low soft phonation (b) and high loud phonation (c). Subject a female 36 years of age. At 34 years of age had acute purulent rhinitis and osteomyelitis of left maxilla and palate. This progressed to necrosis and sequestration of bones on nasal septum, all of palate and superior alveolar ridges. In successive surgical procedures over period of 13 months sequestra and fragments of all of palate, superior alveolar ridge and nasal septum were excised. Process clinically inactive at time of this study. Note progressive medialward motion of the patient's left torus tuberius, and underlying mass of levator muscle, with increasing effort of phonation. As the lax and irregularly shaped palate is raised, dimples appear at the sites of insertion of the levator muscles.

pharyngeus muscle was represented by only a sparse distribution of fibers in this fascial plane, and in one specimen no elements of this muscle could be identified. This muscle extends downward into the lateral wall of the oropharynx, where it was inconstantly distinguishable as a vertical band lying medial to the pharyngeal constrictor and posterior to the pharyngopalatinus, with which it is commonly fused inferiorly.

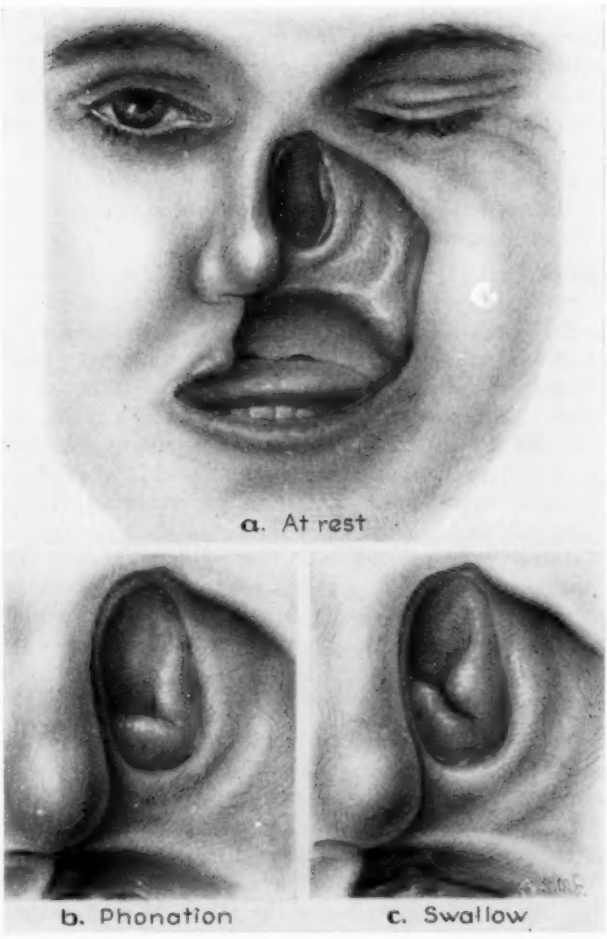

Fig. 8.—Pharynx of subject C. P. at rest (a), and in position of low soft phonation (b) and of swallow (c). Subject an adult male, two years previously had radical maxillofacial excision for carcinoma of the left maxillary antrum. The left side of the nose, adjacent maxilla, floor of the orbit and eye were removed. Note the simultaneous adduction of the torus and appearance of the mass of the levator muscle incident to phonation and swallowing.

THE LEVATOR MUSCULATURE

Cinematic Studies. The area of the nasopharynx was exposed by maxillofacial surgery in four subjects who were available for these studies (Figs. 5, 7, 8, 9). In each instance there was an optunity to observe the upward movement of the palate in the nasopharynx and the coincidental medialward movement of the medial end of the posterior wing of the tubal cartilage. In three of these subjects the motion of the palate and of the oropharynx could be observed simultaneously.

The optimal exposure for purposes of this study was that of subject H. H. (Fig. 5) in whom the nasal septum, inferior conchae and the major portions, of the hard palate and the superior alveolar ridges had been removed. The posterior margin of the hard palate was retained, providing a normal firm anterior attachment for the soft palate. The pharynx of this subject at rest is illustrated in Figure 5a and it is noted that the palate is partially pendant from its base, the pharyngopalatine folds are relaxed in curved contours, and the medial edges of the tubal cartilages are barely visible in the lateral nasopharynx. With the effort of high phonation (of the sound "ah-h-h") the palate rises so that its dorsal surface is slightly above the level of the hard palate. Synchronously the posterior wing of each of the tubal cartilages moves in a posterior and medial direction from the fixed anterior wing. At this time, the pharyngealpalatine folds move medialward and become flat bands with straight medial borders.

Incidental to the brief act of swallowing these motions are exaggerated. For a period of 0.05 to 0.10 seconds their position is that seen in Figure 6c, with the palate lifted high and with a new mass, of the levator veli palatini muscle, moving medialward and upward between the wings of the tubal cartilage, causing the posterior wing of the cartilage to move still farther posteriorly and medially. At maximal elevation of the palate the mass of the levator muscle obscures the end of the posterior wing from sight. Simultaneously the pharyngopalatine folds converge still farther.

These motions are schematically represented in Figure 6, drawn from a dissected specimen which had been transected at a transverse plane approximating the anterior margin of the retained hard palate of this subject, H. H. The levator muscles and tubal cartilages are shown in perspective as their positions were modified in simulation of the observed motion of the exposed pharynx.

In subject G. R. (Fig. 7) the extent of excision was similar to that in H. H. (Fig. 5) with the additional removal of the entire

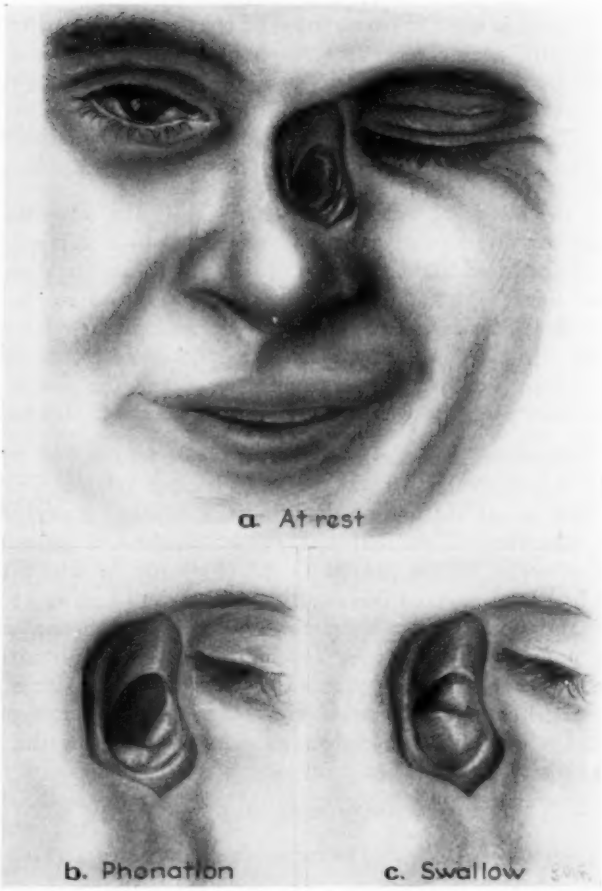

Fig. 9.—Left nasopharynx of subject B. C. at rest (a) and in position of phonation (b) and swallow (c). Subject an adult male who underwent excision of area indicated, including the adjacent orbit and the eye, incident to removal of carinoma in ethmoid sinus one year before this study. An external orifice 2.5 x 3.5 cm in size remained (see Figure 9-A). The patient had no disability of speech, but habitually wore a plastic obturator occluding this orifice for cosmetic effect. Note the simultaneous adduction of the torus and appearance of the mass of the levator in views "b" and "c."

hard palate and adjacent septum. The soft palate, thus deprived of its fixed base, is lax and pendant. The left wall of the nasopharynx is visible and during the effort of phonation (Fig. 7 b and c) the medial part of the posterior end of the tubal cartilage and the mass of the levator move in a manner identical to that seen in patient H. H. The palate, being lax, shows marked irregularity of its ventral surface, with variable dimples at the sites of levator insertion.

In the third subject of this series, C. P., the left side of the nasopharynx and the adjacent soft palate has been exposed by excision of the hard palate, the nasal conchae, and the lateral nasal wall on that side (Fig. 8). This area was also exposed in the fourth subject, B. C., by extensive excision in the area of the ethmoid sinus (Fig. 9). In each of these subjects, the motions of the dorsal aspect of the palate, the levator veli palatini mass, and the medial end of the tubal cartilage are those described in subjects H. H. and G. R.

The movement of the salpingopharyngeal fold and its contained muscle was concealed by the adjacent levator veli palatini or the soft palate in all but one of these subjects having surgical exposure of the pharynx. This fold was visible, incident to phonation, in the oropharynx of the patient O. B. (Fig. 10) in whom the soft palate had been resected unilaterally. With the pharynx at rest, its lateral and posterior walls remained smooth in contour, posterior to the position of the excised faucial pillars. With phonation, the salpingo-pharyngeal fold appeared as a vertical bar rising medialward and slightly anteriorward from the lateral-posterior pharynx. This bar was not separately distinguishable at or above the level of Passavant's ridge.

THE CONSTRICTOR MUSCULATURE

Anatomical Studies. The constrictor musculature (Fig. 11) encloses the cavity of the pharynx, the contours of which are determined largely by current contents. This musculature is basically lamellar in pattern and is fused with the dense pharyngeal aponeurosis which comprises its inner lining. The constrictor musculature is variable in prominence and distribution, particularly as it joins the vertical musculature to accomplish the variety of motions of the palate and to control the palato-pharyngeal sphincter and the oropharyngeal isthmus. The greatest variability is found in the anterior origin of the superior constrictor. The portions arising from the hamulus and pterygomandibular raphe differ considerably in degree in different cadavers. There is also a variable degree of accentuation of the superior constrictor muscle in the lateral and posterior pharyngeal walls at the level of the palate. The degree of variability of the

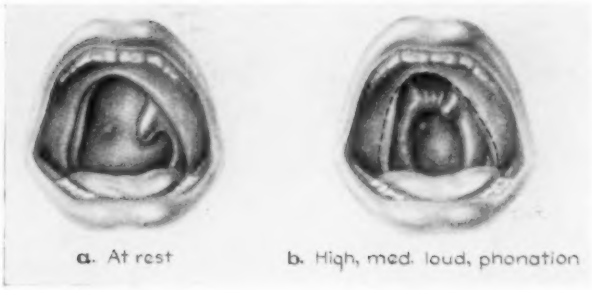

Fig. 10.—Pharynx of subject O. B. at rest (a) and in position of high and medium loud phonation (b). Subject a male, 71 years of age. One year before this study partial surgical resection of the right palate, including the faucial pillars, the soft palate and a small portion of the posterior edge of the hard palate on that side was performed. Currently, he commonly wore a prosthesis, with the aid of which he was able to swallow normally and had but a slight nasal quality of speech. Without this prosthesis, he had a moderate nasality of speech and frequent nasal regurgitation of liquids. Note that incident to phonation the left palato-pharyngeal fold and the leftward displaced uvula are drawn upward and medialward. Simultaneously there appears a broad puckered transverse bar (Passavant's ridge), at the level of the elevated uvula, and the right postero-lateral angle of the oropharynx is filled in by a vertical mass in the area of the salpingopharyngeus muscle.

muscle in this area is difficult to evaluate by reason of the variety of positions of the pharynx at the time of death. In some cadavers (e.g. Fig. 4a), the agonal position is that of contraction of the constrictor, forming a horizontal ridge demarcating the naso- and oro-pharynx, particularly accentuating the lateral recess of the nasopharynx (fossa of Rosenmüller).

Cinematic Studies. The cinema studies which were particularly useful in demonstrating the action of the constrictor musculature were those made upon an adult woman, H. F., having a wide unrepaired cleft of the palate (Fig. 12) and two older men, O. B. and C. G., who had undergone partial excision of the palate (Fig. 10, 13) incident to removal of local neoplasms.

One of the most striking and unexpected actions observed was that of the medial motion of the paired hemiuvulae in the subject having an anomalous cleft of the palate (Fig. 12). This is illustrated in two positions assumed by the hemipalate incident to phonation in Figure 12b and 12c. The extent of this medialward motion varied much with the current particular effort of phonation and on one occasion of gagging the tips of the hemi-uvulae were but 3-4

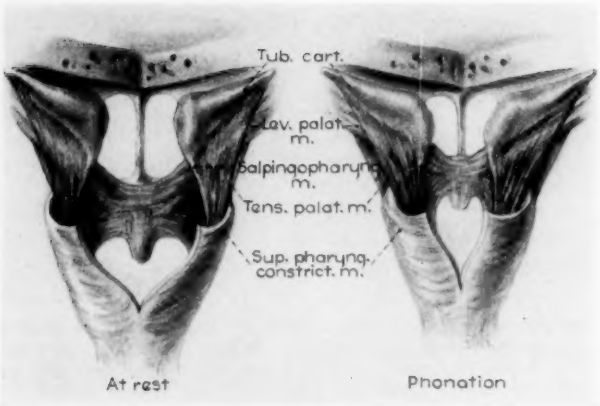

Fig. 11.—Artist's drawing of position and relations of musculature of anterior pharynx, viewed from behind with posterior wall opened and spread.

mm apart. On each of these occasions the hemi-uvulae were moved independently and more actively than the hemi-palate at their base. During the more extreme of these medial motions the two pharyngopalatine folds could be seen to arrange themselves as two limbs of an incomplete "V" with medial ends concealed lower in the pharynx and lateral ends at the hemi-uvulae. The glossopalatine folds remained in a more lateral position. In subject C. G. (Fig. 13) similar medialward motion of the uvula and single pharyngopalatine fold was also noted, persisting after partial excision of the palate.

Simultaneously with the motions of the palate in these subjects during phonation there appeared a prominent fold on the posterior pharyngeal wall (Passavant's ridge) and on the lateral wall (see Figs. 10, 12, and 13). In subjects H. F. and C. G., this fold was seen to be at the level of the bases of the uvulae. It was prominent in subject H. F. and less prominent in the two subjects having partial and total resections of the palate respectively. The appearance of this fold was associated with abrupt and proportionate elevation of that part of the posterior-lateral pharyngeal wall below this level.

On careful inspection at different gradations of intensity of phonation in these subjects it was found that the medially directed motion of the lateral pharyngeal wall occurred at a lower intensity of phonation than did the anteriorward motion of the posterior wall.

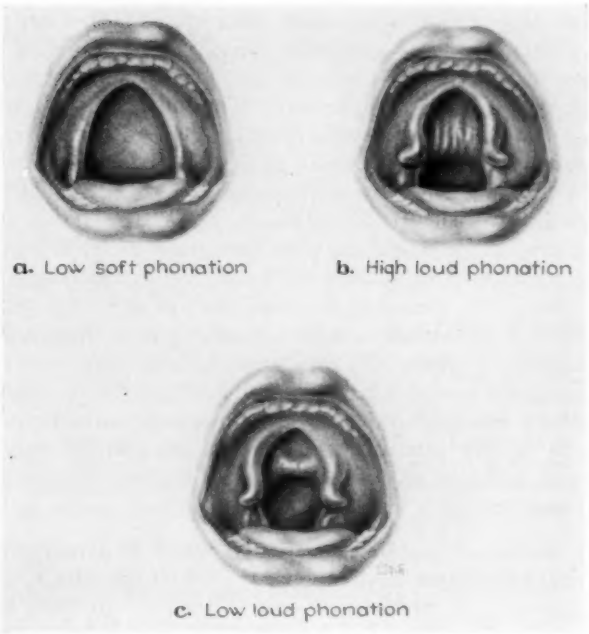

Fig. 12.—Pharynx of subject H. F. at rest (a) and in position of high loud phonation (b) and low loud phonation (c). Subject is a woman 33 years of age. She had an extensive anomalous cleft of the soft and hard palate, upon which no surgical repair had been attempted. Despite the fact that she wore no prosthesis, she had no apparent difficulty in deglutition and her speech, though nasal in quality, was remarkably comprehensible. Note appearance of Passavant's ridge and medialward motion of hemi-palate and palatopharyngeus folds incident to phonation.

With increasing intensity of phonation, the prominence of this ridge on the lateral and posterior walls increased progressively.

COMMENT

These studies have well illustrated the profit of comparing static cadaver anatomy with kinetic anatomy in living patients with surgical or anomalous exposure. The extent of anatomical variations in the area of the pharynx are familiar to all who have dissected this organ. These may be classified into (1) variations of musculature within standard muscle-fascia arrangements, such as differences in level of the insertion of the pharyngopalatine muscle into the constrictor muscles of the lateral and posterior pharyngeal wall and

(2) the relative quantity of muscles and the corresponding prominence of the actions which they accomplish. This variation in quantity has been noted particularly in the muscles within the salpingopharyngeal fold, as illustrated (Fig. 4 b, c, d, e), and in the posterior wall of the upper pharynx. The functional significance of this variation in the salpingopharyngeal muscle is not clear, for we have been unable to observe the isolated function of this muscle. The variation in the portion of the superior constrictor which lies in the posterior wall is probably far more significant, and is probably correlated with the effectiveness of formation of Passavant's ridge and the corresponding medialward projection in the lateral wall of the pharynx. The variations in prominence of this ridge with maximal effort of phonation in high voice have been described in the three subjects in whom this area was observed (Fig. 10, 12, 13). These variations probably have little effect upon the function of the anatomically intact pharynx having a normal innervation. However, it is equally probable that they do considerably qualify the recovery of function of the pharynx after trauma, surgery or neurological impairment.

An additional anatomical variation which is particularly helpful in relating cadaver anatomy and motion of the area is the postlethal persistence of positions assumed in the circumstances of death. This postmortal variation has formed a basis for the extensive study by Lerche of the action of the esophagus and pharynx.[3] Similar variations were noted in the specimens employed in this study, and a specimen demonstrating a pose of action was selected for illustration in Figure 4-A, in which the posterior wing of the tubal cartilage is raised, and the ridge covering the salpingopharyngeus muscle and the lateral extension of Passavant's ridge stand out prominently.

The basic patterns of inter-relation of the motions of the naso- and oropharynx here show have been suggested previously from studies in the cadaver with inference of their significance in function by Wardill,[5, 6, 7] and others.[4, 8, 9] These investigators have been concerned particularly with function of the pharynx as related to speech. The fine gradations of motions are best seen in speech, in which the upper pharynx continuously modulates the basic tone of voice provided by the larynx. The extent of mobility is best seen in deglutition, in which the levator and constrictor muscles contract maximally. The pattern of motion in the upper pharynx in speech and in swallowing are generally similar. This motion is summarized in the two composite illustrations of the palate and pharynx (Fig. 14) in which the mobile structures of the area of pharyngopalatine closure are drawn in a framework of the cranium transected horizontally

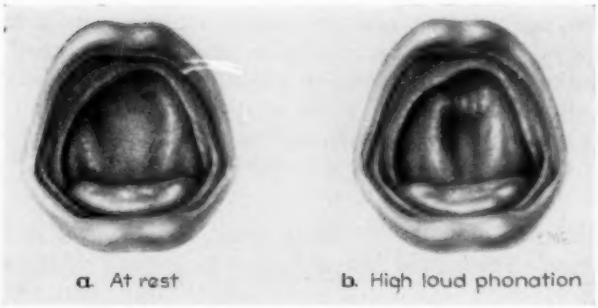

Fig. 13.—Pharynx of subject C. G. at rest (a) and in position of high, loud phonation (b). Subject a male 70 years of age. Three weeks before this study he had an extensive resection of the central portion of the soft palate and the posterior portion of the hard palate. At the time of the study, he had slight nasal regurgitation of liquids and a markedly nasal quality of the voice. Note the appearance of Passavant's ridge in position of phonation. This ridge is relatively shallow, compared with that which is seen in subject O. B. (Fig. 10) and is much less pronounced than that in subject H. F. (Fig. 11).

at the level of the mid-nasopharynx. The coordinated motions accomplishing this fundamental act which are here indicated include:

1. The elevation and posteriorward motion of the palate, by the action of the levator veli palatini.

2. The elevation and posterior and medialward motion of the posterior wing of the tubal cartilage by the levator veli palatini and a filling in of the lateral pharyngeal area by the mass of salpingopharyngeal fold suspended from this wing of the tubal cartilage.

3. The anteriorward projection of the posterior pharyngeal wall (as Passavant's ridge) and the medialward projection of its lateral wall.

The degree of inter-relation of these separate actions is increasingly appreciated as these phenoma are observed in motion.

The observations here reported serve to emphasize the importance of the vertical components of motion of the pharynx. This vertical motion is the resultant of multiple muscle actions, including both intrinsic muscles of the pharynx and a number of extrinsic muscles which insert into the hyoid, larynx and tongue, and act, in the human, to raise this integrated "hyoid mass," the palate and the pharyngeal walls, during swallowing and effortful speech. These

extrinsic muscle include the stylopharyngeus, styloglossus, stylohyoideus, mylohyoideus and the anterior and posterior bellies of the digastricus. This multiple suspension of the "hyoid mass" in the neck makes possible the mobility which is required in the swallowing of semisolid material of varied mass and consistency, in the modulation of a variety of sounds, and in the maintenance of an open airway despite great variations in relative position of the head and trunk.

Although all of the previously mentioned muscles play a part in pharyngeal elevation the key intrinsic muscle of the pharynx in this levator mechanism is the levator veli palatini, the motion of which is synchronized in action with that of the pharyngopalatinus and salpingopharyngeus muscles and facilities the function of each of these by elevation of their site of attachment—the soft palate and the posterior wing of the tubal cartilage, respectively. In contrast with the variation in the other muscles of the pharynx, as noted previously, the levator muscle is remarkably constant in site and contour, and it is the principal mover of the soft palate. By symmetrical action of the levators, and the pharyngopalatine muscles which are inserted fanwise into its aponeurosis, the palate is moved upward and posteriorly in a smooth dome incident to phonation. This smooth contour apparently depends, however, upon fixation of the palatal aponeurosis and palatal ligament to the osseous palate, for there is a vector of posteriorward motion in the action of the levator veli palatini and pharyngopalatine muscles. The significance of this fixation is illustrated when one compares the motion of the soft palate during phonation in normal individuals (H. H.) with those in whom this anchor had been removed surgically (G. R. Fig. 7). Without the hard palate attachment the soft palate is lax.

The mechanism which extends the levator action most directly to the posterior orpharynx is that of the mobile posterior wing of the tubal cartilage and the salpingopharyngeus fascia and muscle. The close relation of the motion of the levator veli palatini and that of the posterior wing of the tubal cartilage was described by Wardill[5, 6, 7] and by Simkins.[10] Harrington[4] studied this area cinematically in one subject having a partial exposure of the nasopharynx persistent after a maxillofacial resection and in one subject having an unrepaired cleft palate. The maximum medialward motion in this area which as noted in this latter study, incident to phonation, was approximately 4 mm. The structures comprising the lateral wall at this level were not distinguished.

In the studies here reported, this close relation of the action of the levator veli palatini and the elevation of the posterior wing of

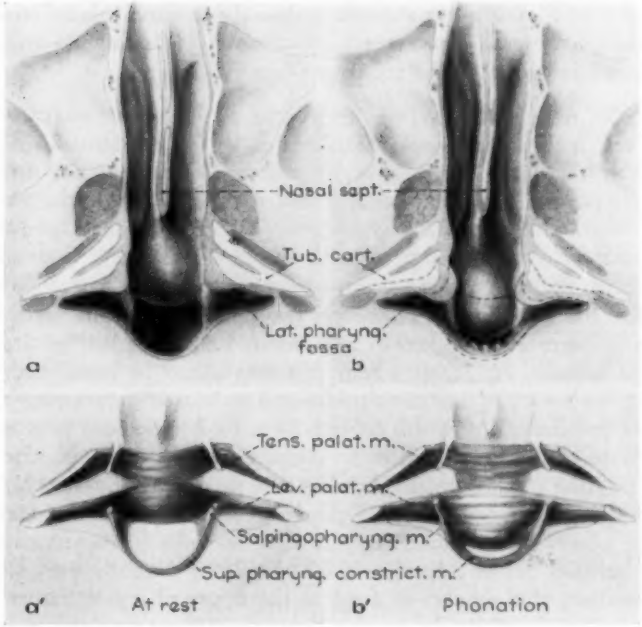

Fig. 14.—Composite drawing of area of palatopharyngeal closure at rest (a) and in position of phonation (b). The relative position of the musculature is separately diagrammed in figures "a" and "b."

the tubal cartilage was noted, and the motions were further found to coincide with those of the pharyngopalatine folds. Furthermore, as these studies include the actions of swallowing as well as of speech the medialward motion of the tip of the tubal cartilage was found to be greater than had been found previously. Here we see the combination of upward motion and adduction comparable to that noted in the action of the pharyngopalatinus. The contribution of this medialward motion to the act of swallowing is probably considerable, for the salpingopharyngeal fascia and muscle must follow the motion of the tubal cartilage in a manner comparable to a spar and its attached sail. As the medial margin of the tubal cartilage moves toward the midline the salpingopharyngeal fascia and muscle must effect an adduction and elevation of the lateral walls of the oropharynx from the level of the palate and downward. The salpingopharyngeus muscle contributes to this adduction and elevation to a variable degree, depending upon the quantity of this muscle within the sal-

pingo-pharyngeus fascia. These observations fail to support the impression of Simkins[10] to the effect that the levator palatini and the salpingopharyngeus muscles act reciprocally, to raise and to lower the posterior wing of the tubal cartilage.

The muscle continuing the levator action in the anterior oropharynx is the pharyngopalatinus and its levator action is distributed generally within the pharynx by reason of its insertion into the greater portion of the lateral and posterior perimeter of the pharyngeal wall. In standard physiological descriptions these pharyngopalatine arches are considered with the glossopalatine arches as constituting the isthmus fauceum—interpreted as a portal between mouth and pharynx.[11] However, the pharyngopalatine folds were found in surprising degrees of adduction in these studies, particularly in the subject, H. F., in whom this area could be more easily observed by reason of her cleft palate and associated broadening of the whole oropharynx. In this subject these folds were seen to converge nearly to the midline incident to gagging and, furthermore, the relative position of their medial borders was that of an inverted V, with its convergence in the inferior-posterior portion of the oropharynx. These observations would suggest that the pharyngopalatine folds may actually act as a barrier which wholly or partially prevents the penetration of the bolus of food to the upper pharynx, rather than as laterally placed guides to this bolus as it penetrates the pharynx. This interpretation is made more probable by the observation of Negus[2] that the stream of food is diverted from the midline by the epiglottis and passes down the lateral portions of the pharynx.

If the pharyngopalatine folds act during swallowing as a dorsal and posterior barrier to the stream of food, they would supplement the action of the palate, superior constrictor and salpingopharyngeus muscles in preventing penetration of the food into the nasopharynx. This role of the pharyngopalatine muscles as supplementary mechanisms in the closure of the palatopharyngeal isthmus is mentioned by Negus in his extensive review of the comparative anatomy and function of the pharynx and larynx.[2] His interpretation of the action of these muscles is apparently based upon homologic comparisons. The pharyngopalatine muscles may contribute also to the adduction of the lateral pharyngeal wall at the level of uvulopharyngeal closure by their supplementation of action of that part of the superior constrictor muscle in which they are intermixed as they are inserted into the palate, as pointed out by Strong.[12] This supplemental mechanism of pharyngopalatine closure would be particularly useful if the function of the palate were impaired neurologically, as by paralysis of the levator veli palatini, or anatomically,

as by cleft palate. This mechanism may well contribute to modulation of speech and swallowing in normal and in anatomically or neurologically impaired individuals.

The familiar combination of motions of elevation and adduction which have been noted are found also in the superior constrictor muscle in its sphincter action in the posterior and lateral walls of the pharynx at the level of pharyngopalatine closure. The anteriorward and medialward motion at this level has been well-demonstrated in three subjects, in one of whom this area was exposed by anomaly and in the others by excision of the palate. It is noteworthy that this ridge was most prominent in the subject who had the cleft palate, in whom this mechanism of closure was particularly useful. The appearance of this ridge was associated with a symmertical elevation of the lateral and posterolateral aspects of the pharyngeal wall below this level. It is probable that this elevation is an effect of the combined action of the vertical salpingopalatinus and the obliquely placed pharyngopalatinus muscles, for at this level in the posteropharyngeal wall the fibers of the superior constrictor muscle lie in the transverse plane or at angles slightly divergent from it. Thus, they contribute effectively to the palatopharyngeal sphincter, but cannot be expected to raise this portion of the pharyngeal wall.

SUMMARY

The anatomy and function of the musculature of the upper pharynx has been studied by dissection in 18 cadavers and by cinematic analysis of movement in subjects in whom this area was exposed by anomaly (1 patient) or by surgical excision of adjacent face or palate (6 subjects). The position and action of the levator veli palatini, pharyngopalatinus, salpingopharyngeus and superior constrictor muscles have been studied, and a number of inter-relations of their action identified. The commonly observed action of the pharyngopalatinus, salpingopharyngeus and superior constrictor muscles was their combined action of elevation and constriction of the upper pharynx. The levator veli palatini muscle was identified as a prime mover in this combination of action of the intrinsic muscles of the pharynx.

The author is indebted to Mrs. Louise Marshall Follett, medical artist, of the Department of Medical Illustration; and Mr. James McCarron, motion picture photographer of the Division of Visual Education, University of Minnesota. The author also wishes to thank Mrs. Jean Hirsh Fish, Head, Department of Medical Illustration, Dr. Edward A. Boyden, Professor and Head, Department of Anatomy, Dr. Logan Leven, Clinical Associate Professor, Department of Surgery, University of Minnesota and Dr. Chester Swinyard, Professor, Department of Anatomy, University of Utah, for their encouragement, assistance and criticism.

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VII

TINNITUS AURIUM IN NORMALLY HEARING PERSONS

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As considered here, tinnitus aurium is a medical term describing sounds of physiological or pathological origin, which may or may not always be perceived in consciousness.

Kerrison enumerated five general groups of sounds: 1) obstructive, 2) circulatory—vascular alterations, 3) labyrinthine—cochlear sounds, 4) neurotic—instability of the auditory nerve, 5) cerebral sounds—involvement of the auditory centers.

Fowler has divided tinnitus into two categories: 1) vibratory, mechanical, exogenous—factual sounds within the body, and 2) nonvibratory, biochemical endogenous—total absence of sound without the body.

Vibratory tinnitus is real sound of a physical source such as muscle activity, or vascular alteration. Nonvibratory tinnitus is nonfactual sound: an illusion of sound caused by an irritation of the auditory neural elements. The points of origin may be anywhere from the tympanic promontory, along the pathways to the cortex inclusive.

Atkinson also has divided tinnitus into two categories; extrinsic and intrinsic, which appear to include the two types already mentioned. He considers intrinsic tinnitus as an auditory paresthesia, a paresthesia of the auditory nerve, of vascular origin and to be so treated.

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Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions submitted by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Wegel recorded: "Tinnitus is a pathologic symptom . . . I am under the impression that the presence of tinnitus . . . generally indicates an active or progressive lesion and that the cessation of it . . . is an indication that the degeneration or atrophy of tissue has been arrested." But then he continued, "people entirely without tinnitus are extremely rare, if such cases exist at all."

In 1941 Fowler wrote, "It has been found that the presence of tinnitus is always associated with more or less deafness." In 1944 he altered this view writing: "It may be, and often is, present in some form in persons who have no apparent aural or other disease."

Kopetzky stated that tinnitus is a symptom signifying disturbed sensation, a symptom of aural disease. He continued that tinnitus may appear before symptomatic deafness.

Lempert suggested on the basis of his observations associated with middle ear surgery that "tonus impulses originating in the sensory fibers of the trigeminus, the sympathetic, or glossopharyngeal may enter the tympanic plexus, but normally [are] not heard." In selected cases, he recommended tympanosympathectomy.

Fowler further described tinnitus, of which the patient is consciously aware, as "audible," and tinnitus not ordinarily impinging on the consciousness as "subaudible." He found tinnitus in 86% of 2000 patients. He indicated that subaudible tinnitus must be sought for by examination. He also determined that the loudness of tinnitus was within 5 db to 10 db above threshold. Tinnitus may be measured for frequency, loudness and quality. It may be a single frequency or multiple frequencies and difficulties may be encountered in its identification. The exact loudness may also be difficult to determine. Fowler described techniques for making such measurements.

Audible tinnitus at times appears to interfere with hearing. Patients often state that were it not for their head noises their hearing would be better, and that when the head noises are louder the deafness is more severe. It does not necessarily follow that the tinnitus is always responsible for this. Possibly with increased deafness the head noises are less easily masked and so appear louder subjectively. Fowler has described the busy line effect, whereby receptor cells and neural pathways already preoccupied by an intrinsic stimulus are not receptive to an external stimulus.

Some conditions in which audible tinnitus have been observed are:

1) Otosclerosis.

2) Meniere's disease.

3) Lermoyez's Syndrome.

4) Pressure or neuritis of the auditory apparatus; brain tumor, eighth nerve tumor, aneurysm.

5) Otitis media; acute, chronic, suppurative, nonsuppurative.

6) Otitis interna; acute, chronic.

7) Deafness, conductive, perceptive, mixed.

8) Normal hearing with discrete frequency defect.

9) Nasopharyngeal Diseases: Eustachian salpingitis, sinusitis, pharyngitis, mucosal hypertrophy, hyperplasia, tumor, infection of lymphoid tissue.

10) Dental pathology: malocclusion, malfunction of temporomandibular joint, impaction, infection.

11) Myositis; cervical, pharyngeal, tympanic.

12) Intoxication-drug; quinine, alcohol, salicylates, caffeine, tobacco, antiluetic agents, streptomycin, thyroid gland extract.

13) Intoxication-systemic; gastrointestinal, foci of infection.

14) Allergy.

15) Cardiovascular pathology; blood dyscrasias, anemias, hypertension, hypotension, vascular anomalies, arteriosclerosis, cardiac diseases.

16) Metabolic dysfunction; thyroidism, water balance disturbances.

17) Trauma, acoustic, acute.

18) Trauma, acoustic, chronic.

19) Systemic fatigue.

20) Momentary tinnitus, spontaneous (idiopathic).

21) Impacted cerumen.

22) Cervical constriction.

23) Psychoses.

24) Otic herpes.

25) Bell's palsy.

26) Foreign body trauma to the ear.

27) Head injury, concussion, postconcussion syndrome.

28) Myringitis.

29) Hemorrhage, tympanum or myringa.

The ideal approach to the treatment of audible tinnitus would seem to be a therapeutic assault on the related etiological factors. At present there is no sure way to accomplish this. Frequently the etiological agent no longer exists, however the tinnitus persists. Some of the contemporary measures are:

Medical:

1) Medication; bromides, barbiturates, other sedatives, potassium iodide, vitamins, benzyl cinnamate, antiallergic drugs, histamine therapy, intravenous procaine.

2) Local therapy to disease processes.

3) Elimination of drugs and intoxicants.

4) Elimination of foci of infection.

5) Correction of faulty gastrointestinal function.

6) Correction of metabolic diseases.

7) Control of diseases of the vascular system and blood forming organs.

8) Dietary control of fluids, salt, and water balance.

9) Dental rehabilitation.

10) Intratympanic medication.

11) Therapy directed to correct nose and throat pathology, including roentgen and radium therapy.

12) Politzerization, inflation, message.

13) Removal of cerumen.

14) Psychotherapy.

15) Hearing aid.

16) Electrical therapies, i.e., ultra violet, quartz lamps, galvanism.

Surgical:

1) Otologic
   ossiculectomy
   mastoidectomy
   tympano-sympathectomy
   fenestration of the labyrinth
   obliteration of the saccus endolymphaticus.

2) Rhinologic.

3) Spinal tap.

4) Cranial surgery for tumor, vascular anomalies section of eight cranial nerve.

5) Splanchnectomy and similar technics for alleviation of hypertension.

TABLE I.

REPORT OF SOUND EXPERIENCED BY 80 NORMALLY HEARING SUBJECTS, IN A SOUND-PROOF ROOM AND INCIDENCE OF TINNITUS IN 100 PATIENTS WITH DEAFNESS.

	NUMBER OF HARD OF HEARING PATIENTS	PER CENT OF HARD OF HEARING PATIENTS	NUMBER OF NORMAL SUBJECTS	PER CENT OF NORMAL SUBJECTS
Sound heard (tinnitus)	73	73	75	93.75
No Sound (no tinnitus)	27	27	5	6.25
	100	100	80	100

Fowler has emphasized the value of explaining to the patient the nature of his tinnitus: that it is a symptom and not a disease, and that despite its annoying and distressing presence, it does not imply a threat to him. An understanding of the symptom and a recognition of its relative significance in some instances may reconcile the sufferer to his burden.

From the foregoing it appears that both audible and subaudible tinnitus have been described as a symptom associated with impaired hearing, or with systemic diseases, and yet they have been observed in the presence of these same factors, in persons considered healthy. The implication has been made that tinnitus may be an early symptom preceding impaired hearing.

It has been noted that healthy persons with normal hearing have reported tinnitus when the ambient noise level is low. The opportunity presented itself to us to determine the incidence and character of subaudible tinnitus by exposing normally hearing persons to an environment in which the ambient noise level was considerably less than in ordinary living conditions.

Two separate groups of persons were studied: normally hearing, healthy, adults who experienced tinnitus rarely or not at all, and hard of hearing adults, veterans of military service.

A sound-proof chamber was used. The ambient noise level was probably between 15 db and 18 db (re: 0.0002 dynes per cm^2). Exact measurements could not be made due to the limitations of the sound level meters at hand.

In a previous study we investigated the influence of tinnitus of the doughnut type of receiver worn over both ears. Normally hearing persons were placed in the sound proof chamber and the tinnitus experienced with and without receivers was compared. They were unable to determine any difference and we concluded that the ambient noise of this chamber was not an intruding factor.

Eighty adults, apparently normally hearing males and females, from 18 to 60 years of age were included. The selection was predicated on a denial of past or present aural disease. They reported no deafness or tinnitus, and considered themselves in good health. They were representative of a sedentary population, including physicians, dentists, teachers, students, administrators, clerks and housewives.

Upon entering the sound-proof room the subjects are instructed to make notes of sounds which might be detected. No suggestion was given that the source of sound might be within the subject himself. The time of observation was usually limited to five minutes or less. Written details of their observations were obtained.

One hundred hard-of-hearing patients, consecutively admitted to the Clinic, composed a control group. Their histories, otorhinological examinations and pure-tone audiograms were obtained. A diagnosis of deafness, its type, and the presence or absence of tinnitus and its description were recorded. See Table I.

A total of 39 different sounds were described by both groups. Of these, 27 sounds were named in the impaired group, and 23 sounds were named in the normal group. The sounds described as "buzz," "hum," and "ring" were enumerated most frequently in both groups, comprising at least 50% of the responses of each group. Eleven sounds recorded were identified in both groups.

TABLE II.

DIFFERENT SOUNDS DESCRIBED BY BOTH GROUPS.

		NUMBER OF PATIENTS WITH IMPAIRED HEARING	NUMBER OF NORMAL SUBJECTS
1.	Bell	3	0
2.	Buzz	12	13
3.	Drone	1	0
4.	Hiss	3	3
5.	Hum	10	16
6.	Ring	32	11
7.	Steam	4	0
8.	Roar	5	2
9.	Whistle	9	3
10.	Click	3	0
11.	Tap	1	1
12.	Falling water	3	4
13.	Heart beat	2	0
14.	Truck	1	0
15.	Rushing	1	0
16.	Airplane	2	1
17.	Singing	1	0
18.	Insects, crickets	2	6
19.	Fog horn	2	0
20.	Musical sound	1	0
21.	Machinery	1	0
22.	Rumble	1	0
23.	Hollow sound	1	0
24.	Squeal	1	0
25.	Echo	1	0
26.	Surf	0	1
27.	Pressure	0	2
28.	Vibration	0	1
29.	Squeak	0	3
30.	Throbing	0	1
31.	Rustling leaves	0	1
32.	Stuffiness	0	1
33.	Tunnels	0	2
34.	Pulse	0	7
35.	Rubbing cloth	0	1
36.	Watch tick	0	2
37.	Thumping pulsation	0	4
38.	Zooming-whizzing	1	2
39.	Sea shell	2	0

TABLE III.

NUMBER OF SOUNDS REPORTED BY EACH 75 NORMAL SUBJECTS WITH SUBAUDIBLE TINNITUS AND BY EACH OF 75 HARD OF HEARING PATIENTS WITH AUDIBLE TINNITUS.

	NUMBER OF PATIENTS	PER CENT OF PATIENTS	NUMBER OF NORMAL SUBJECTS	PER CENT OF NORMAL SUBJECTS
1 sound	48	64	54	72
2 sounds	20	27	15	20
3 sounds	6	8	3	4
4 sounds	1	1	2	2
5 sounds	0	0	1	0.75

The number of different sounds described by the normally hearing group and by the patients with deafness, in terms of percentage, are in general agreement. While a majority in both groups reported hearing only one sound, a substantial number of persons in each group distinguished two or more sounds.

TABLE IV.

DIAGNOSIS OF DEAFNESS AND INCIDENCE OF TINNITUS IN 100 PATIENTS.

DIAGNOSIS	NUMBER OF PATIENTS	TINNITUS PER CENT OF 100 PATIENTS
Conductive deafness without otosclerosis		
Tinnitus constant	3	
Tinnitus inconstant	10	13
No tinnitus	7	
Total	20	
Otosclerosis (1 case mixed deafness 7 cases conductive deafness)		
Tinnitus constant	4	
Tinnitus inconstant	4	8
No tinnitus	0	
Total	8	

TABLE IV.—(Continued)

DIAGNOSIS	NUMBER OF PATIENTS	TINNITUS PER CENT OF 100 PATIENTS
Perceptive deafness		
Tinnitus constant	21	
Tinnitus inconstant	18	39
No tinnitus	16	
Total	55	
Mixed deafness		
Tinnitus constant	2	
Tinnitus inconstant	5	7
No tinnitus	1	
Total	8	
Mixed deafness of one ear, perceptive deafness of other		
Tinnitus constant	3	
Tinnitus inconstant	1	4
No tinnitus	1	
Total	5	
Diagnosis not available		
Tinnitus constant	2	
Tinnitus inconstant	0	2
No tinnitus	2	
Total	4	

Of this group, 73% of the patients experienced tinnitus, 27% did not. All the eight patients in this series with deafness due to otosclerosis had tinnitus. Of a larger group of otosclerotics in our Clinic, 83 patients, 85% have tinnitus and 15% are free of it.

COMMENT

Seventy-three per cent of 100 unselected patients with deafness described tinnitus as a symptom. Twenty-seven per cent experienced no tinnitus.

Audible tinnitus was experienced by 94% of the 80 apparently normally hearing adults when placed in a testing situation having an ambient noise level no greater than 18 decibels, re: 0.0002 dynes per cm^2. It appears that tinnitus is present constantly but is masked by the ambient noise which floods our environment. This ambient noise level for ordinary quiet living conditions usually exceeds 35 db, and apparently is of sufficient intensity to mask physiological tinnitus, which remains subaudible.

It would appear, then, that tinnitus will not be eliminated by any treatment but at best can only become subaudible. This, of course, would be welcomed both by the patient and the physician.

CONCLUSION

The kinds of head noises described by patients with impaired hearing as a symptom associated with their deafness and those sounds described by normally hearing healthy adults, elicited while in a sound-proof room, appear to be similar.

These head noises seem to occur in about the same order of frequency.

Tinnitus, which is subaudible, may be a physiological phenomenon in an intact auditory apparatus.

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VIII

NASAL CYSTS AND THE FACIAL CLEFT CYST THEORY

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The purpose of this paper is to re-introduce a theory which lately received little consideration. It may help in evaluation of every kind of cyst on and around the nose, with the establishment of a logical classification and a proper nomenclature. It also might serve as an incentive to further investigations.

The following considerations are chiefly devoted to the cysts protruding from the insertion line of the nose. They are not mucoceles, nor are they of the odontogenic type: they are soft part cysts. Histologically, they are mostly of mucoid nature with a minority showing dermal developmental structure.

The prototype of this group of cysts is one which arises immediately underneath the ala nasi. It grows in three directions: toward the nasofacial fold, toward the vestibule of the mouth and toward the vestibule of the nose. It can invade secondarily the nasal cavity beneath the anterior third of the inferior turbinate; usually, it lies in a groove of the bone and is movable. Its lining shows the structure of respiratory epithelium or of a modification of the latter: variations of this sort are characteristic of this cyst. Ciliated cells are often found while the number of goblet cells is very variable. The content is serous, mucous or a combination of both. Cholesterol was never found in it.

As to the origin of these cysts two theories might be quoted, one considering them as the result of obstruction of a glandular duct, the other as the late effect of some early disturbance during the embryonic development of the nose. One is the Retention Cyst Theory and the other the Facial Cleft Cyst Theory.

The first to report glandular retention cysts underneath the nasal ala were Zuckerkandl (1882) and Chatellier (1892), and Brown Kelly presented his retention cyst theory in 1898. The latter studied sagittal sections through the mucocutaneous junction of the

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Read at the meeting of the New England Oto-Laryngological Society, in Boston, June 18, 1952.

vestibular floor and saw that the membrane was formed almost entirely of glandular tissue. On approaching the ala, the glands increased in number. Several times the ducts were dilated in a cyst-like manner. In spite of these findings no connection was claimed between glands and a true cyst. Neither did Klestadt[22] or Doering report a similar relation in sections cut through both: cyst wall and nasal mucous membrane. But such a connection should follow from the retention cyst theory.

It is difficult to understand on the basis of the findings of Brown Kelly why all these typical cases are reported as lying underneath the ala. It sounds equally strange when he describes the upper parts of the ducts as an intermediate between skin and mucous membrane with all the reports showing clear-cut mucoids. These are obstacles to the application in case of the subalar cyst.

However there are good reasons why this theory should not be refuted completely. First: glandular retention is seen everywhere where glands occur. Second: studying, in the collection of Kallius, 34 human embryos, I was able to find regularly anlages of glands in the region under consideration; they are generally recognized as the origin of cysts (hamartoma). Further investigation should include the floor, medial to the ala.

In the above mentioned studies, I found a tube-like epithelial protrusion on the lateral edge of the floor which showed a tendency to form a cystic choristoma.

The retention cyst theory prevailed until 1913. In this year a new point of view was presented (Klestadt[17]) according to which the subalar cyst could be compared to the branchial cleft cysts. A developmental concept was considered by Brown Kelly, but discarded by him. Later, Blumenthal (1913) thought of a similar origin when he described a recurrent abscess in the ala of the nose. This was considered to be a purulent cyst though without proof as to its cystic nature. The articles of Brown Kelly and of Blumenthal were unknown to me at the date of the publication (Klestadt[17]) in which the term: *facial cleft cysts,* was suggested.

The facial cleft cyst theory, considering its present status, is supported by the following arguments:

1. The area where the subalar cyst is found is identical with that of the fusion of the three embryonic facial processes.

2. The epithelial lining of the cyst shows the character of genuine nasal epithelium.

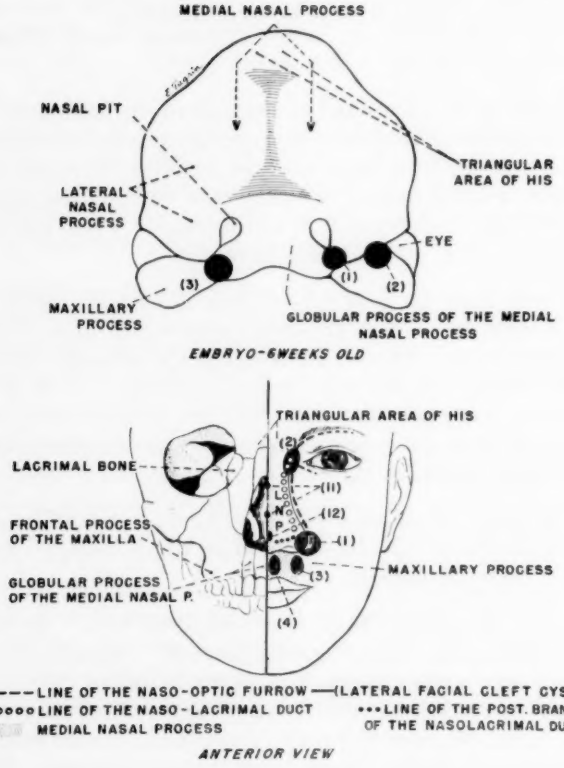

Fig. 1.—Position of the Developmental Cysts of the Nose. 1. Subalar facial cleft cyst. 2. Nasoethmoidal facial cleft cyst. 3. Globulomaxillary or lip and palate cleft cyst. 4. Same as 3, only named "Premaxillary cyst." 11. Fistulae leading to median superior cysts of the nose. 12. Cyst of the Jacobson gland.

3. Analogy with the branchial cleft cysts is apparent. Against this it can be argued that islets of cells were not yet found in this particular area, neither in embryonic life nor later. However, this objection could be applied equally to branchial cleft cysts, yet nobody has dismissed the branchiogenic theory on this basis. Phylogenetic considerations help to explain the lack of cell islands of the kind mentioned here. During normal intrauterine life no actual fissure appears in this area, as it would fall into the line of the naso-optic furrow or of the branchial arches. This way the conditions do not favor the formation of cell inclusions. On the other hand when similar clefts do exist, epithelial islands appear not infrequently. An intermediary situation arises when shifting, folding and pressing occur together with volume changes in the vicinity of fissures. A good example is furnished by the papilla palatina as nests of epithelial cells are found inside the latter quite often (Peters, Rawengel, Rydzek).

4. Symmetrical occurrence is not infrequent and is better explained by developmental disturbance than by retention.

5. Occurrence in a father *and* a daughter (Kasche) points definitely to a hereditary factor.

6. The indentation underneath the subalar cyst should be considered as an aplasia and not as the effect of erosion or pressure. Opinion in favor of pressure was stressed but never verified histologically. Physical considerations speak against a pressure effect as the cyst fluid exerts the same pressure in all directions. However, in the case of the subalar cyst it would act more powerfully in a circumscribed area and would affect the most resistant tissues of the vicinity. On the other hand it is known that bone defects are often associated with tumors in areas in which pathological cleft formation occurs. This without doubt is so in the case of cysts. The lacrimal bone may be absent, replaced by a naso-ethmoidal cyst (Klestadt[20]); there may be a large bone defect underneath an anterior median maxillary cyst (as seen in consultation with Dr. Albert Weiser, Fall River, Massachusetts); or diastases may be present through which fistulae run ending in dermal cysts in the midline of the nose (Holmes, Benjamins, Klestadt[21]). Further evidence was furnished by a symmetrical agenesis of the frontal processes of the maxillary bones (Klestadt[18]), (called by me submuco-subcutaneous facial cleft fissures), and this even in the absence of a cyst. These processes are placed in the line of the lateral insertion of the nose, this line again being a potential area of developmental cysts.

7. Two cases were reported in which the subalar cyst showed a protrusion against the lateral line of the nasal insertion. This line

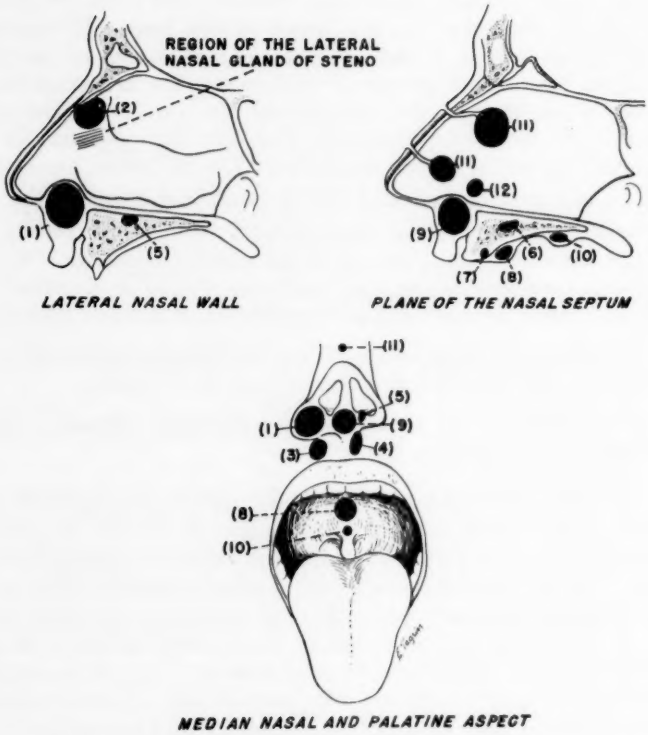

Fig. 2.—Position of the Developmental Cysts of the Nose. 1. Subalar facial cleft cyst. 2. Nasoethmoidal facial cleft cyst. 3. Globulomaxillary or lip and palate cleft cyst. 4. Same as 3, only named "Premaxillary cyst." 5. Nasopalatine cyst of the nasal floor. 6. Intermediate nasopalatine cyst. 7. Cyst of the incisive foramen (i.e. oral end of the nasopalatine tracts). 8. Cyst of the palatine papilla. 9. Median anterior maxillary cyst. 10. Median posterior palatine cyst. 11. Fistulae leading to median superior cysts of the nose. 12. Cyst of the Jacobson gland.

corresponds to the embryonic naso-optic furrow. The authors (Uffenorde, Huizinga) considered their findings as supporting the facial cleft theory.

Between 1920 and 1930 further observations were reported showing that similar cysts may appear within the entire line corresponding to the naso-optic furrow. Vogel collected from the literature two cases regarding the middle section of this furrow. Two have been described (Klestadt[20]) as occurring in the naso-ethmoidal border region. Comparable cysts were reported further upwards, in the eyelids. It appears that the entire area where under pathological conditions an oblique facial cleft may exist could be considered as a field of potential cleft cyst formation. It is remarkable that underneath the alae only mucoids were observed, in the intermediate section of the lateral insertion line of the nose only epidermoids, dermoids and penetrating fistulae, while both occur in the cranial portion. Rare but remarkable are the keratinizing epidermoids in and around the topmost corner of the orbit, claimed generally to be mucoceles. The basis for this diagnosis is not too strong: but interpretation as facial cleft cyst is justifiable as pathological clefts can reach as high as this point.

Brüggemann[6] presumed that the subalar cyst originates from a prolongation of the nasolacrimal duct into the ala. But this anterior branch occurs only in lower animals; in man one merely sees traces of a posterior branch represented by short grooves running towards the incisive canal. Thus a cyst of similar origin would be first, an atavistic structure and second, it would be located behind the nasal aperture and would be able to invade the vestibulum only secondarily. Being nearly a cyst in theory it could not be considered as a true facial cleft cyst.

Cysts deriving from a regular nasolacrimal duct were never reported. The reason might be its very recent development as against the naso-optic furrow. At the period of its formation the most violent developmental forces are petering out. This argument is even more valid in the case of the problematic posterior branch.

It is unfortunate that these cysts are reported under a multiplicity of names. Handouza Bey rightly emphasized the desirability of a uniform nomenclature. I think that the main term should designate the genetic origin and topographical and other characteristics should be added to it. An example shall be given by discussing one synonym of the subalar facial cleft cyst giving, afterwards, a short survey of the entire field of the developmental cysts on and around the nose.

The synonym in question is: Nasoalveolar cyst. It offers only a topographical characterization. Thus it could mean any cyst within the line from the insertion of the columella to that of the ala; several possibilities offering themselves.

A glandular retention cyst of the mucocutaneous junction could be confounded with some type of cyst which originates in and between the fusion lines of the primitive palate. The "globulomaxillary cyst" of Thoma offers an example. This is a topographical term; but at the same time it reports precisely the origin of the cyst in the tissue uniting the globular protrusion of the median nasal process and the maxillary process. The name could be as well "lip and palate cleft cyst." It is interesting that their behavior corresponds to that of the clefts of the same name inasmuch as their location is independent in relation to the teeth. The nomenclature causes difficulties only in case the cyst is located between incisors or supernumerary teeth, and regular teeth; they were then labelled "premaxillary cysts" by Roper-Hall.

There are many types of cysts expanding within the nasoalveolar region. They can be formed here in connection with configuration changes of the infranasal area, of the medial nasal process or around the primitive palate. Here belong a cyst of the oral end of the naso-palatine tract lying in the opening of the foramen incisivum (the opening of the nasopalatine canal); the cysts of the papilla palatina, and a cyst underneath the insertion of the columella, called the median anterior maxillary cyst.

All the enumerated cysts which might be found in competition with the subalar facial cleft cyst regarding the term "naso-alveolar cyst" belong to a median caudal group of developmental cysts. The subalar cyst itself is a member of the lateral group which we have already discussed.

The median caudal group contains furthermore all the median posterior palatine cysts located in the line of union of the palatinal protuberances of the maxillary processes. This is a large group of mucoids; their lining shows as a rule the structure of the oral squamous epithelium. Surface epithelia of the line of closure, and nests of epithelial cells in the posterior part of the palatinal papilla are credited to be their cells of origin (Bergengrün, Rydzek, Rawengel).

Concerning the median caudal group considerable knowledge was contributed by the anatomist Meyer and by dental researchers (Thoma, Schroff, Stafne et al) after 1930.

The last group of developmental cysts is a median cranial group in the midline of the dorsum nasi. It consists of epidermal and dermal cysts with their stalks (Holmes, Benjamins). The mechanics of the origin of all midline cysts remains a difficult problem (Klestadt[21]).

The cranial region is a kind of meeting ground for cysts originating from the closure of the anterior end of the neural canal and its appendices (Grünwald[9]). The median anterior maxillary cysts are situated near the point of origin of another developmental type, the cysts of the organ of Jacobson (Sieur and Jacobs; Kelemen). Finally it should be mentioned that in front of the anterior head of the middle turbinate the lateral nasal gland of Stenon is found and that it was claimed as the origin of facial cleft cysts. It may give rise to endonasal cysts.

There is a great variety of cysts on and around the nose related to the developmental process. They have to be differentiated from each other as well as from other types of cysts. They have to be classified and brought under an adequate nomenclature. This work will have to be undertaken in the future. The problem is of interest to the rhinologist, the ophthalmologist, the dentist, the general surgeon as well as to the anatomist and embryologist. Elaboration of the facial cleft cyst theory offers a new field of study. Cases of facial cleft cysts are rare but not to such a degree as to justify the silence with which they are met in almost every textbook of otolaryngology.

38 Rock Street.

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IX

PRIMARY CARCINOMA OF THE MASTOID PROCESS

CASE REPORT

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Among the earliest reports on carcinoma of the middle ear and mastoid process is that of Schwartze,[1] which appeared in 1775. Since then there has been a total of two hundred or so reported cases, of which perhaps half could be authentically considered as being primary to the mastoid process or middle ear, or the so-called intrinsic type.[2, 3] Of this group the squamous cell carcinoma variety is the most frequent,[4] while adenocarcinoma is the rarest.[5]

That cancer primary to the mastoid process and middle ear is still extremely infrequent is evident from Table 1. At this hospital only one case has been seen since 1940, during which interval there have been 116528 admissions to all in-patient services of the hospital. The more recently reported glomus jugulare tumors have not been included in this report, nor have they appeared in any of the cases summarized in the table below. Owing to the usual location of glomus jugulare, we feel that this is really an extra-cranial structure and that the non-chromaffin paraganglioma to which it may give rise invades the middle ear and mastoid process secondarily.

These figures show a wide variation in the incidence of this type of pathology, depending largely on whether the basis for the report is:

(1) The over-all registration of patients for a general service institution;

(2) The number of registered patients in an institution devoted primarily to tumor work; or

(3) The number of patients seen on a specialized service; e.g., the otological service of an E. N. T. department.

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TABLE 1.
FREQUENCY OF OCCURRENCE OF INTRINSIC CARCINOMA OF THE MIDDLE EAR AND MASTOID PROCESS.

Institution	No. of Patients	No. of Cases of Intrinsic Carcinoma	% Intrinsic Carcinoma Cases	Ratio Case Per No. Patients	Reported By
Univ. of Michigan Hospital	40,000	2	0.005	1: 20,000	Furstenberg[5]
London Hospital	200,000	1	0.0005	1:200,000	Tod[6]
Roswell Park Memorial Inst.	70,000	10	0.014	1: 7,000	Mattick and Mattick[7]
Jefferson Hosp. Otology Service of Dr. H. J. Williams	1,883	3	0.16	1: 625	Towson and Shofstall[8]
Manhattan Eye Ear & Throat Hosp.	212,000	5	0.0023	1: 50,000	Robinson[9]
University Ear Clinic (Basal)	45,000	6	0.013	1: 7,690	Thorell[10]
Wilkes Barre Gen. Hosp.	1,200	1	0.08	1: 1,200	Buckman[16]
Ohio State Univ. Hospital	116,528	1	0.0008	1:116,526	

Carcinoma intrinsic to the mastoid process and middle ear constitutes 1.5% of all tumors of the ear,[7] and there is general agreement among the authors that it occurs mostly after 40 years of age, with equal frequency in males and females, and is extremely rare in Negroes.[8, 4] Statistical deductions from so few authentic cases as listed above can at best be but questionably valid.

REPORT OF A CASE

Case: J. A.

History: The patient, a white male, age 52 yrs., physician, gave a history of periods of deafness of the right ear with an accompanying sensation of fullness in the same region for the past 2½ years. These symptoms were associated with a variable degree of pain, which, however, was readily controlled by aspirin. Periodic treatment with penicillin resulted in complete remission of symptoms from time to time, only to become worse later. The middle

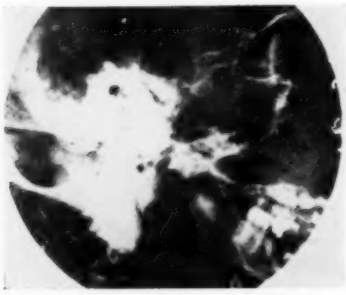

Fig. 1.—Mastoid area, 3 months pre-operatively. Note moth-eaten appearance of mastoid roof with malignant changes.

ear did not become permanently impaired until three months prior to his admission to this hospital, and at that time (October, 1951), x-ray studies revealed extensive erosion of the right-mastoid bone (Fig. 1). Two weeks before entering the hospital he developed a marked right facial paresis, and it was this turn in events which overcame his resistance toward entering the hospital, and which modified a rather fatalistic attitude toward his illness. He was admitted to University Hospital on January 17, 1952. Interesting in the history, although perhaps not directly related to the topic of this report, are his incapacitating intervals of chronic arthritis extending over many years; an episode of coronary thrombosis in 1938, and a cholecystectomy in 1939. At no time had there ever been any drainage from either ear.

Findings: The patient, a well developed, but not well nourished individual, presented a firm smooth swelling over the right mastoid process, causing that area to appear much more prominent than the left. Tenderness to moderate palpatory pressure was found over his swollen area and the skin was readily movable over it. The lumen of the external auditory canal was quite patent to within 3-4 mm of the tympanic membrane, and because of occlusion beyond this point, the status of the middle ear could not be ascertained at this time. No enlarged regional or cervical nodes were palpable. An almost complete paralysis of the facial muscles was present on the right side. T. 99.0°; P. 90; R. 20; Blood Pressure 142/90. Routine blood and urine laboratory work were within normal limits, and the blood serology was negative. Roentgenological studies done here upon admission showed positive evidence of a neoplastic invasion of the mastoid process. At all times the left ear appeared normal.

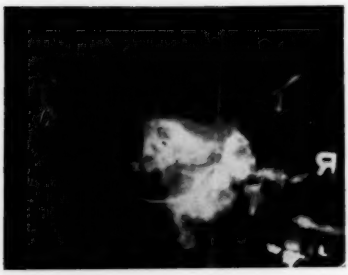

Fig. 2.—Post-operative x-ray film. Note boundary of bone removal.

Clinical Course: With a pre-operative diagnosis of neoplasm of the right mastoid process, an extensive radical mastoidectomy was done on January 19, 1952. Using the post-auricular approach, a sub-periosteal, smooth, mushroom-like mass of firm encapsulated tissue was found, measuring 3.5 cm in diameter by 1.0 cm in thickness. This accounted for the raised mastoid area as noted above. The "stem" of the neoplasm had eroded from within through the bony cortex in the upper mastoid region. The pathological diagnosis from frozen sections of this material was, "Low grade squamous cell carcinoma, well differentiated." Further progress of the operation disclosed that the new growth had intermittently broken down mastoid bone with extensive involvement of the contiguous dura. The wall of the lateral sinus was also found to be widely covered with neoplastic growth reaching downward toward the jugular bulb. This widespread infiltration necessitated the removal of the entire mastoid tip and inner wall of the mastoid process upwards to the base of the petrous pyramid and forward to the facial canal. After exposing the facial nerve, the inner table of the mastoid bone was removed until the posterior and superior semi-circular canals were skeletonized. It is questionable whether the invasive process had extended to the petrous tip, or beyond our visual field in the zygomatic area, even after skeletonizing the glenoid fossa and removing the bony anterior wall of the external auditory canal (Fig. 2). Sero-sanguineous exudate found in the field of operation was reported sterile after culturing it. No metastic lesions were found. It was felt that extensive removal of infiltrated tissues wherever encountered would facilitate subsequent irradiation of the more deeply situated structures, and accordingly the post-auricular flap was not closed. The large subjacent tympano-mastoid cavity was packed loosely with iodoform gauze, preparatory to the implanting of radiation sources

Fig. 3.—Plastic wheel for carrying radiation sources of Cobalt 60.

directly into this cavity. The latter was accomplished on the fourth post-operative day by Dr. Arthur G. James, who placed into the cavity a plastic wheel carrying six point sources of Cobalt 60,* delivering a total radiation dose of about 7000 r in four days, at which time it was removed (Fig. 3, 4 and 5—Courtesy of Dr. Arthur G. James).

On the eleventh post-operative day the tympano-mastoid cavity was closed by suturing the original post-auricular flap in place, after making a large communicating orifice between the external auditory canal and the cavity to facilitate post-operative care.

The patient withstood all of the above procedures well, the major post-operative complaints being that of transitory vertigo with nausea, and some nystagmus, all of which gradually wore off.

The pathological tissue diagnosis confirmed the previously examined frozen sections as being "well differentiated squamous cell carcinoma with invasion into bone" (Fig. 6).

Post-Operative Diagnosis: Carcinoma, primary to the right mastoid process, with extensive contiguous invasion.

Follow up x-ray studies on the seventeenth post-operative day showed, in addition to an extensive defect of the squamous portion of the temporal bone extending back to the occipital bone and up-

*Furnished by the Atomic Energy Commission and Prepared by Dr. Joseph L. Morton.

Fig. 4.—Plastic wheel in situ for irradiation of the post-operative tympano-mastoid cavity with six radiation sources of Cobalt.

ward into the parietal area, a further defect of the inner portion and entire tip of the mastoid process down to the middle ear (Fig. 2). The facial paralysis of the right side became complete following the primary irradiation reaction. The patient was discharged from the hospital February 11, 1952, on the twenty-second post-operative day.

During the ten months that have intervened since his operation, the patient has had a rather stormy course, suffering considerable head pain corresponding mostly to the trigeminal nerve distribution. Severance of the 5th and 9th cranial nerves and the first three cervical nerves has been suggested for relief of this pain.

Discussion: We have felt that this case has several points of interest:

1. Absence of aural discharge. While some investigators have reported cases of carcinoma of the mastoid process and middle ear as having no discharge from the external auditory meatus, and although, in Figi and Hempstead's series of 38 cases, only 11 had otorrhea, nevertheless, most writers give the presence of long standing chronic discharge as a most common finding.[2, 3, 7, 11, 12, 13, 14] That the irritating effect of chronic drainage might cause cancer, had been frequently advanced,[11, 15] and is still mentioned, though much in a historical light, by practically all the more recent writers. There had never been any discharge from the ear of our patient, and so, obviously, this could have no bearing on the etiology of cancer in this instance.

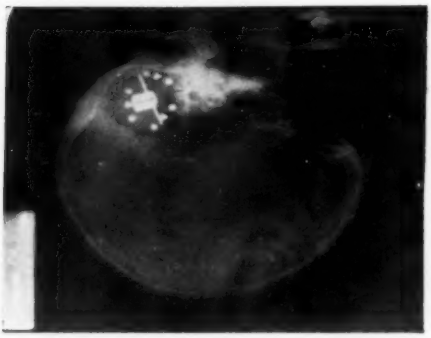

Fig. 5.—X-ray picture of radiation wheel in place.

2. Intact tympanic membrane. By virtue of finding an intact tympanic membrane plus a neoplasm of the mastoid process and middle ear cavity, this tumor is felt to have been primary to the mastoid process, and to have subsequently extended into the middle ear cavity at a later stage.

3. Pain. Pain is so frequently a major symptom in a case as advanced as this one, that the relatively minor role it played here preoperatively was deemed worthy of note.

4. Cobalt 60. Adjunctive treatment with an artifical radioactive isotope, Co. 60, appears to be rather unique in this situation, for we have found no like use of it in the literature for this type of case.

Summary: A very brief resume of the incidence of intrinsic cancer of the mastoid process and middle ear has been presented, along with the report of an authentic case to be added to a relatively meagre literature. This case was felt to be unusual because of the following combination of circumstances:

(1) Absence of aural discharge.

(2) Intact membrana tympani.

(3) Minor role of pain (preoperatively) in an advanced case.

(4) Adjunctive treatment with an artificial radioactive isotope, Co 60.

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X

A CONTROLLED COMPARISON OF THE EIGHTH NERVE TOXICITY OF STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

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A strong clinical impression that dihydrostreptomycin neurotoxicity has a predilection for the auditory apparatus, whereas streptomycin mainly affects the vestibular apparatus, has existed for some time. A multitude of articles has appeared in the literature on the subject, subsequent to the original description by Brown and Hinshaw on the neurotoxicity of streptomycin.[1]

In 1951 an opportunity was presented to compare the relative toxicities of these drugs in a controlled experiment.* The primary objective was to note the relative therapeutic effects on pulmonary tuberculosis; as a corollary, toxicities were carefully checked.

Patients with pulmonary tuberculosis,† in some instances with additional tuberculous lesions, were considered for streptomycin or dihydrostreptomycin therapy at a conference of tuberculosis specialists. Those patients considered suitable for chemotherapy were alternately assigned to one of two groups. One group received drug "A," the other drug "B." One drug was streptomycin sulfate, the other dihydrostreptomycin sulfate.‡ Which was "A" and which was "B" was not known to any of the physicians in charge.

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*Similar studies were carried out simultaneously by seven other groups of investigators in other sections of the United States.

†At the Weimar Joint Sanatorium, Weimar, California.

‡It appeared obvious by February, 1952, that drug A had predominantly auditory toxicity, whereas drug B had primarily vestibular toxicity. Although still a secret at that time to all locally, the clinical feeling was that "A" must be dihydrostreptomycin, and "B" streptomycin. This later proved to be correct.

Each patient received one gram daily of either drug "A" or "B" for a total of 120 days. In rare instances the exigencies of a particular case required modification of this regimen. Para-amino-salicylic acid was given concomitantly, usually 12 grams daily in three or four divided doses. A lesser amount was given to patients unable to tolerate this dosage, but not less than eight grams daily.

A total of 34 patients received drug A; it was dihydrostreptomycin sulfate. 34 patients received drug B; it was streptomycin sulfate. Of those on drug A, 24 had far advanced tuberculosis, 9 moderately advanced, and one minimal disease. Of those on drug B, 28 had far advanced disease, 3 moderately advanced, and 3 minimal.

Detailed medical records were maintained for each patient. Special note was made of sputum bacteriology, x-ray changes, temperature trends, body weight and neurotoxic manifestations. The neurotoxic manifestations were studied both by subjective report of the patients and by objective tests.

Objective vestibular and auditory tests were routinely done, with few exceptions, 1, prior to therapy, 2, at the conclusion of the 120 days of therapy, 3, two months post-therapy, 4, four months post-therapy, and 5, six months post-therapy. A few patients have been followed even longer. More frequent tests were done when a change of status was suggested clinically.

Hearing was checked by pure tone audiometry, using a new, calibrated audiometer. Two carefully trained, exceedingly competent technicians made all the audiograms. Significant variations were checked by the otologist (W. E. H.).

Vestibular responses were treated by the modified Kobrak method[2] using 5, 10 or 30 ml of ice water as the stimulus. Results were tabulated on special cards noting the quantity of ice water used, the type of nystagmus, the duration of the latent period, the duration of active nystagmus, the character of the nystagmus, its degree, and the subjective sense of vertigo.

The first patient was started on therapy in January, 1951. The last of the series started in November, 1951, and finished the 120 day course in February, 1952.

THERAPEUTIC EFFECTS

Careful study of the clinical status of the patients in this program, by the criteria mentioned earlier, failed to reveal any convincing therapeutic advantage of either streptomycin or dihydrostreptomycin over the other, in the therapy of tuberculosis.

TABLE 1.

OTOTOXICITY.

DRUG	VESTIBULAR DISTURBANCES (NO. OF PATIENTS)		AUDITORY DISTURBANCES (NO. OF PATIENTS)	
	MILD	MODERATE	MILD	MODERATE
Dihydrostreptomycin (A) 34 patients	2	0	4	1*
Streptomycin (B) 34 patients	4	2	0	0

*One additional patient, not included in the series, suffered an auditory disturbance of moderate degree. He was a ten year old boy (C. L.) with miliary tuberculosis and meningitis. He was first seen in a critical condition, and received streptomycin for one month before starting on drug A (dihydrostreptomycin). Because of the far advanced disease, he received two grams of drug A daily during most of the 120 day course. His ear, nose and throat structures were normal except for hypertrophied tonsils. Six months post-treatment he had a grossly apparent hearing loss, the audiogram showing drops of 20, 50, 55 and 60 decibels at frequencies 1,000 to 8,000 c.p.s. Audiograms made 8, 10 and 12 months post-treatment showed no improvement. The Rinne test was bilaterally positive. Urinalyses were normal. His body weight varied from 70 to 100 pounds during treatment. He is excluded from the series because of the very large doses received (approximately 60 milligrams per kilogram of body weight daily) and because of the nature of his disease which may have damaged the central nervous system.

The therapeutic goal, as defined prior to treatment in each case, was completely attained in 59% (20 cases) of patients receiving drug B (streptomycin), partially attained in 38% (13 cases) and a complete failure was noted in one case. Drug A (dihydrostreptomycin) was more consistently successful, the therapeutic goal being completely attained in 79% (27 cases), partially in 18% (6 cases) and there was failure in one case (3%). These differences do not appear to be statistically significant in a series so small.

OTOTOXICITY

Of the 34 patients who received dihydrostreptomycin, only two evidenced any manifestation of vestibular disturbance. It was subjective only, consisting of transient vertigo of minimal degree. Auditory disturbances, however, were evident in five patients, mild in four, but moderately severe in one (Table 1).

Of the 34 patients on streptomycin, six had vestibular disturbances, four of mild degree, two moderately severe. No auditory disturbances occurred in this group.

AUDITORY DISTURBANCES

Those cases in which there were auditory disturbances, all five of the patients receiving dihydrostreptomycin, deserve closer scrutiny. Four had disturbances of mild degree only, that is, a drop of 20 decibels or more at 8,000 cycles per second, compared to pretreatment thresholds. In each case the impairment became manifest *after* completion of the course of treatment, the earliest two weeks post-treatment, the latest five months post-treatment. Once manifest, the drop became greater in two of the patients over a two-month interval. The maximum change was 40 decibels.

The patient (T. D.) on dihydrostreptomycin who had a moderate hearing loss was a 21 year old man with far advanced pulmonary tuberculosis. His ear, nose and throat structures were normal except for some chronic tonsillitis. At the end of treatment a hearing loss was clinically apparent and demonstrable by audiogram. Five months post-treatment it had progressed so that at 500 c.p.s. there was approximately a 10 decibel drop, at 1,000 c.p.s. a drop of 30 decibels, and at 2,000, 4,000 and 8,000 c.p.s. a drop of 40 decibels, compared to the pre-treatment thresholds (Fig. 1). He had worked in a noisy lead mill for one year.

VESTIBULAR DISTURBANCES

As mentioned above, only two patients of those on dihydrostreptomycin had any manifestation of vestibular disturbance, and then only of a minimal, transient, subjective nature.

Of the six patients on streptomycin who showed vestibular disturbances, four had only slight difficulty. This was characterized by subjective vertigo, i.e. a false sense of motion, more or less throughout the course of therapy. It ceased on discontinuance of the drug.

Two of the patients on streptomycin had vestibular disturbances of moderate degree. One was an eighteen-year-old girl (G. C.) with far advanced pulmonary tuberculosis. Four weeks after completion of therapy there was no response on caloric stimulation with 5, 10 or 30 ml of ice water in either ear. Three months later the responses were almost normal and five months later there were completely normal responses to minimal (5 ml) stimulation.

The second patient (M. H.) was a 21-year-old man with far advanced pulmonary tuberculosis. Again four weeks post-therapy

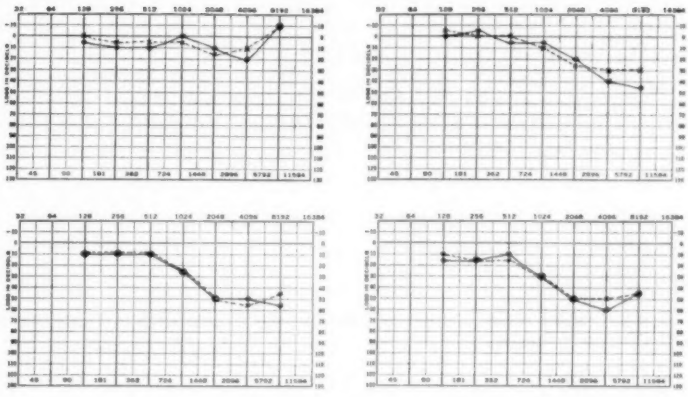

Fig. 1.—Audiograms of patient T. D. showing progressive post-therapy hearing loss (Drug A—dihydrostreptomycin). (X—A.C left ear; O—A.C. right ear)

Audiogram No. 1—Pre-therapy.
Audiogram No. 2—At end of therapy.
Audiogram No. 3—Three months post-therapy.
Audiogram No. 4—Five months post-therapy.

no labyrinthine responses could be elicited by caloric stimulation, yet 3 and 5 months later the reactions had returned to normal.

COMMENT

It is evident from this study that the neurotoxic effects on the eighth nerve by streptomycin are definitely and predominantly upon the vestibular apparatus. Equally definite is the predominant neurotoxicity of dihydrostreptomycin on the auditory apparatus.

It may be postulated that the vestibular dysfunction is reversible because the apparatus is an older, more primitive mechanism. The newer, exceedingly delicate auditory apparatus, on the contrary, has little recuperative ability.

SUMMARY AND CONCLUSIONS

A controlled experiment, comparing the therapeutic and neurotoxic effects of streptomycin and dihydrostreptomycin in patients with tuberculosis, was performed.

No distinct therapeutic advantage was found for either drug.

The predilection for vestibular toxicity by streptomycin, and auditory toxicity by dihydrostreptomycin, was established.

Such vestibular dysfunctions become manifest soon after the streptomycin is started, and are reversible, at least with a 120 day course of one gram daily, on stopping the drug.

The auditory dysfunctions become apparent only after 3 or 4 months of dihydrostreptomycin therapy, are progressive for several months even though the drug is stopped, and are apparently irreversible.

The factor of reversibility may hinge on the primitive, resistant nature of the vestibular apparatus as against the newer, delicate auditory mechanism.

The interested assistance of L. Siemion and G. Knudsen, audiometrists, is gratefully acknowledged.

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XI

FOREIGN BODIES AND ROENTGEN EXAMINATION OF PERFORATION OF THE ESOPHAGUS

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The number of patients having a foreign body in the esophagus, seen in this clinic, increases year by year. Causes: 1.) the patients are directed to the clinics by their doctors sooner than formerly; 2.) perhaps people eat more carelessly in these hurried times; 3.) after the war more fish is being consumed in the Netherlands. Fishbones are swallowed remarkable often by the people with false teeth. The fishbone is the most frequent foreign body in the esophagus, here. Among the last 246 cases there were 95 fishbones, 45 small bones, 31 coins, 20 dental protheses, 17 pins and 38 other things.

On the other hand the number of external operations decreases more and more in proportion. This is sufficiently obvious from the following survey:

	NUMBER	EXTERNAL OPERATIONS	CASUALTIES
1907 - 1924	24	3	4
1924 - 1940	125	6	...
1940 - 1952	246	6	...

The number of external operations has decreased strongly, owing to better endoscopic technique and to chemotherapy and the antibiotics.

An external operation is necessary when the foreign body cannot be removed with the esophagoscope. This has not been necessary in Groningen for many years. The indication of late has been perforation of the esophagus accompanied by inflammation. If the foreign body is still in the esophagus it is usually removed by esophagotomy. This however is seldom necessary.

From various handbooks one gets the impression that but two treatments are possible in these cases: esophagoscopy or external esophagotomy. This is probably due to the fact that formerly the

laryngologist was as a rule only well-acquainted with the endoscopic technique. If an indication for an external operation existed the patient was referred to the general surgeon, who removed the object by external esophagotomy. Until today the idea is still maintained that there is only this one alternative. That is not the case, however. Other possibilities have already been very well described by Schlemmer in his survey in the handbook of Denker and Kahler and by others, but they do not seem to have become of common knowledge.

In general an external operation was performed when, in case of a perforation of the esophagus, there clearly appeared symptoms of inflammation. In such a case the perforation must be laid free and care should be taken for ample drainage of the periesophageal tissues, through a collar mediastinotomy according to the Vienna school. This done, the question may again be put how to get rid of the foreign body at the smallest risk. In some cases it will prove best to open the esophagus and take out the object, as was formerly done here in the case of a safety-pin. But in other cases the object may very well be removed by esophagoscopy as soon as possible after the external operation. The big advantage has been that no great opening in the esophagus need be made, and that the leaking, later on often so disturbing, can be avoided.

In the case of some patients, who were admitted with an unmistakable periesophagitis caused by a fishbone, the esophagus was first laid free and then the fishbone removed by esophagoscopy.

In the case of a patient previously described,[1] the line of action differed. It concerned a man of 28 with a big dental prothesis in the esophagus, who had experienced a perforation somewhere else by esophagoscopy. There was a strong emphysema of the neck. The patient had a temperature of 39.7.° The prothesis had got stuck behind the larynx. The esophagus was laid free. Below the prothesis a small opening was made in the esophagus, through which the forefinger might be inserted. The prothesis was gently loosened and pushed upwards and could then easily be removed with the laryngoscope.

In case of periesophagitis x-ray examinations by means of lateral views of the neck is of the greatest importance. X-ray verification takes place regularly in the radiological institute (Prof. S. Keyser) in all cases with few exceptions during and after esophagoscopy.

On the lateral film we see in the first place the reaction of trauma as well of the foreign body, as of the esophagoscopy. This reaction, edema and swelling, caused by inflammation, manifests itself as a widening of the tissue shadow between spine and trachea. It is clear

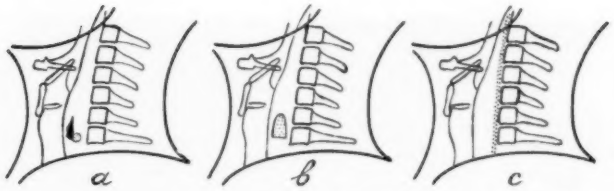

Fig. 1.—Diagrams of three possibilities in perforation of the esophagus.

that it is of importance to ascertain by x-ray check whether the symptoms increase or decrease.

It is still more important that by diagnosing air, mostly between trachea and spine, a perforation of the esophagus can be ascertained. This is the symptom of Minnigerode,[2] which was fully discussed in an earlier paper.[3] Normally no air can be seen between the spine and the trachea. Under pathological circumstances three possibilities may offer themselves (Fig. 1): a.) There are to be seen one or a few small air-bubbles behind the trachea. This need not prove that the esophagus-wall has been perforated. In the case of foreign bodies, which, as is well-known, usually stay in the mouth of the esophagus, one can imagine a few air-bubbles easily adhering to the object between the folds of the swollen mucous membrane, so this air is still in the lumen of the esophagus. b.) On the film a big air-bubble can be seen, which is sometimes outlined below by a sharp clear line, remaining horizontal in various attitudes, in which case we have to do with an abscess with a fluid-level, and above it air from the esophagus-perforation or gas formed by bacteria. As often as not this abscess is located in the periesophageal tissues, but exceptionally it may be found in the esophagus-wall below the submucosa. It will be readily understood that a big bubble principally occurs when the process is gradual. There is an abscess with a tendency to encapsulation. c.) There is an emphysema of the interstitial tissue in the neck. This is a sure sign the esophagus has been perforated. On the lateral films this is usually seen as a light streak in front of the spine, which may reach as high as the nasopharyngeal cavity. So this symptom has the same meaning as the feeling of emphysema in the neck. The great importance of x-ray examination is that in some cases only in that way can the air be shown.

Formerly the point of view was held that if in this or any other manner, a perforation of the esophagus could be established, the operation should be undertaken from the outside. This radical standpoint was pretty universally accepted, especially on the author-

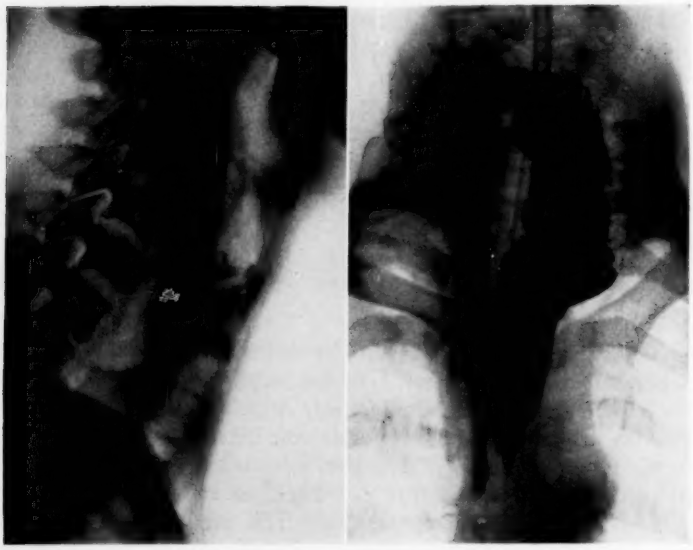

Fig. 2, Case 1.—a. Lateral and b. dorsoventral roentgenograms.

ity of Killian. It had already been well known for a long time that some cases had healed spontaneously, but attention was drawn to the fact that operating in time is better, as one is otherwise liable to be surprised by the so much dreaded mediastinitis. This is certainly very true. By means of the x-ray examination it proved, however, in these cases that a perforation of the esophagus repeatedly healed without further complications.

Diagnosing a perforation of the esophagus was in itself not yet an indication for operating. Circumstances decided. For a time it was an absolute indication if inflammatory symptoms occurred (fever). It became even more difficult with the advent of chemotherapy and especially the antibiotics. Certain cases of periesophagitis were promptly cured in that way. We have also seen examples of this.

On the other hand x-ray examination often gives a hint for immediate operation. In the collection there are a number of x-ray films of patients brought in with large periesophageal abscesses with perforation of the esophagus by foreign bodies. All patients recovered after external operations.

It is remarkable that by a neck operation patients may be saved, in whom the suppuration had already dropped to the mediastinum;

Fig. 3, Case 2.—Dorsoventral roentgenogram.

it is really a collar mediastinotomy. The following patients are examples.

CASE 1.—A woman of 64 was referred to us with the following history: She very likely swallowed a foreign body three days before, while eating soup. Twice esophagoscopy was performed, and the laryngologist thought he saw a foreign body. It could not be removed however.

During the second attempt at extraction he had the impression that a perforation had been caused. The patient received chemotherapy, the temperature hovered between 38° and 39,° but there was a swelling of the neck, especially on the right side, where also an emphysema could be felt. On the x-ray film (Fig. 2a) this emphysema is clearly visible all along the spine. Besides, the periesophageal tissue was much swollen; there was a big air-bubble in it. Not a moment's doubt existed but that the esophagus had to be laid free. A large quantity of purulent matter appeared; behind the esophagus there was a very large abscess cavity, reaching far down into the thorax. A feeding catheter was inserted. When, after a few days the patient did very well, a film was made with a lipiodol tampon in the abscess cavity (Fig. 2b). This reached nearly to the sixth thoracic vertebra.

CASE 2.—A woman of 68 swallowed a fishbone two days before. She had a pain at the lower left side of the neck, and also some pain on pressure. The temperature was 38.1° when she was brought in. Later it proved that on each of the films a fishbone could be seen. The neck was very short, the fishbone lay nearly entirely in the shadow of the clavicle. The fishbone was not found by esophagoscopy, but there was plainly visible an injury to the mucous membrane. The following day the

Fig. 4, Case 3.—Lateral roentgenogram.

temperature rose to 38.5°; there could be felt no emphysema, but on the x-ray film the symptom of Minnigerode was very clear. The shadow of the tissue between the spine and the trachea was very much wider. The following days the temperature dropped, did not any longer rise above 38° and usually hovered between 37.4° and 37.8.° Improvement was noted on the films. The swelling went down and the air disappeared. So we waited for six days, until the films showed swelling again and a bubble with a clear circumference could be seen behind the larynx. The esophagus was then laid free. Again a large abscess was found. There was a wide cavity behind the esophagus, with fibrinous coating, which reached down as far as the aorta. On the last film the fishbone was discovered. After the external operation esophagoscopy was done and the fishbone removed.

The patient did very well. After a few days another film was taken with a lipiodol tampon. The enormous abscess cavity immediately strikes us again in this dorsoventral film (Fig. 3), and it must be taken into consideration that in reality the cavity was still larger because it was not quite filled with the tampon. This cavity stretched downwards as far as the fourth thoracic vertebra.

It is our experience that the perforation of the esophagus is, in the majority of cases, caused by attempts at extraction by esophagoscopy. It has been suggested that the presence of air in the periesophageal tissue was also solely a result of the esophagoscopy. Normally the esophagus is closed, but when the mouth of the esophagus is opened by the esophagoscope air may easily be sucked in. Besides this there also existed an opinion that possibly gas was formed by

certain bacteria. In the following case no esophagoscopy at all was done.

CASE 3.—A man of 53 had swallowed a fishbone a week ago. He got a pain in the neck, the neck began to swell, he could no longer swallow. He was ill, and his temperature on admission was 39.2.° In Figure 4 an imposing lesion may be seen. There is a pronounced widening of the tissue between the trachea and the spine. Here a great number of large air bubbles are to be seen. At collar mediastinotomy a large abscess was found with very much ill-smelling pus.

Our point of view proves to have changed several times during the course of years. Diagnosing the right indication has really become more and more difficult. A hard and fast rule cannot be given. One can surely no longer say that every perforation of the esophagus must be operated upon. Nor even that every perforation with inflammatory symptoms must be operated upon. The indication must be given by careful clinical observation. To which end x-ray examination is of the greatest importance. In every complicated case, where but the slightest suspicion of perforation exists, regular x-ray inspection is necessary.

SUMMARY

Statistics of foreign bodies in the esophagus from the Groningen University Clinic are given. Indications for an external operation are discussed, the principal one being periesophagitis.

When the foreign body is still present it is seldom necessary to do an espohagotomy, in most cases a collar mediastinotomy can be performed and the foreign body can then be removed through the esophagoscope.

The symptom Minnigerode is very important for the diagnosis of a perforation of the esophagus.

Regular x-ray examination of the neck with lateral films is of the greatest value as an indication for operation.

ACADEMISCH ZIEKENHUIS.

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XII

INTRANASAL ENCEPHALOMENINGOCELE

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AND

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Removal of intranasal growths seldom presents great technical difficulty. The greatest number of polypoid nasal lesions are removed in the office with little hesitation and it is not always the case that such lesions are biopsied before surgery, or, once removed are submitted for pathological examination. H. Russell Fischer[1] recently reviewed a variety of pathological nasal tissues routinely submitted over a period of a year and a half. The implication from this review is that rhinologists must exhibit more caution in diagnosing apparently benign lesions without biopsy and use more restraint in removing nasal tissues indiscriminately lest their true nature surprise or embarrass us.

Discussed here is a case in point, one in which the excised specimen proved to be an encephalomeningocele.

According to St. Hilaire,[2] the most commonly accepted origin of nasal encephalomeningocele is from an adhesion of the brain anlage to the germinal membrane. Later development of the skull tends to isolate this area from the brain except at the point of origin. Evidence cited is that cerebral hernias commonly begin during embryogenesis and are already developed at birth. In most cases the nasal obstruction can be observed in the first years of life.

Classification in general has been made either in respect to the kind of tissue involved or the anatomical position of the hernia aperture. If the hernia contains cerebral tissue it may be called an encephalocele, but as pointed out by McGillicuddy,[3] it is difficult and unnecessary to arbitrarily separate the brain from its investments and such herniations are more properly termed encephalomeningoceles.

Presented before The Otolaryngology Staff Meeting, Los Angeles County Hospital, December 17, 1952.

In respect to anatomic distribution there are three main groups:

Occipital—into the occipital region, forming an external tumor. These are said to be the most common.

Sincipital—with externally visible tumors of the nose, or forehead, in the medial corner of the eye, or the anterior part of the orbital cavity.

Basal—These developing into the posterior part of the orbit, the nasal cavity, or the epipharynx, and therefore not externally visible. These are of primary interest to the ophthalmologist and rhinologist.

Sub-division of these basal herniations has been made into:

1. *Encephalomeningocele Spheno-Pharyngea*—which leaves the cranium through the sphenoid bone or enters the epipharynx between the sphenoid and ethmoid bones.

2. *Encephalomeningocele Intra-Nasalis*—which enters the nose through the cribriform plate to form an intranasal tumor. From the year 1813, forty-nine of these cases have been collected in the literature; thirty-five by Gisselsson,[4] thirteen by Moore,[10] and the one presented here.

3. *Encephalomeningocele Spheno Orbitalis* — This is traced through the supraorbital fissure into the orbit behind the eyeball. Strandberg,[5] in 1948 collected thirty-one cases of these from the year 1841.

4. *Encephalomeningocele Spheno Maxillaris*—this also passes through the supraorbital fissure but then continues down through the infraorbital fissure into the sphenomaxillary fossa and appears as a tumor on the medial side of the mandibular ramus.

The first published case of intranasal encephalomeningocele appears to be that of Richter in 1813. His patient was a sixty-year old male treated for a large nasal polyp projecting from one nostril. This was extirpated without untoward sequelae. Some months later another polyp presented. In snaring this, a nasofrontal encephalomeningocele was opened and the patient died of meningitis. On post mortem study two pathological openings in the skull were found, one a tract through the ethmoid bone and one through the frontal bone.

Nager[4] reports a six-year old boy seen because of difficulty in nasal breathing. The mother noticed a growth in the boy's nasal cavity at the age of three, which was snared off the following year. At the age of five a recurrence of the tumor was similarly removed,

followed by a cerebrospinal rhinorrhea and subsequently a pneumococcal meningitis. The boy recovered under urotropin, but later, following a recurrence, he died of streptococcal meningitis. At autopsy an opening in the cribriform plate was found through which dura protruded.

Christian Fenger,[6] in 1895 reported the first case to be operated upon and cured. The patient was a 29-year-old male complaining of difficulty in breathing through the left side of the nose since childhood. A tumor was removed from this side of the nose with some relief for a short time, however, the difficulty returned. Examination disclosed another growth reaching from the nasal cavity into the epipharynx. The tumor was soft and compressible. Encephalomeningocele was suspected and a diagnostic puncture was done. Since no fluid was obtained the tumor was excised with a snare. Cerebrospinal rhinorrhea occurred immediately. A resection of the maxilla was done and the stalk of the tumor was located in a dehiscence posteriorly in the cribriform plate. This was ligated and the dehiscence packed with iodoform gauze. There were no further complications reported. Fenger, was the first to point out as an important diagnostic sign a widened nasal root and an abnormal distance between the eyes. His paper is well worth reading.

Certainly more than 49 cases of intranasal encephalomeningocele have been found and may have been reported. Gisselsson[4] states they occur one in thirty-five to forty thousand births. Of these cases however, in only 20 including the case reported did the patient survive treatment and in two of these no surgery was done. Nine of the remaining patients had other developmental anomalies and died shortly after birth. The rest succumbed following surgery from a meningitis either immediately following or from a later upper respiratory infection. Twelve of the survivals may be credited to antibiotics or recognition of the tumor before surgery, leading to a neurosurgical approach.

The importance of preoperative differential diagnosis can be accepted as more than academic. One notes that of the 34 cases cited by Gisselsson only eight patients survived surgery. Gisselsson[4] and Fleisher[7] list several criteria.

1. A nasal tumor found at birth, or symptoms of nasal obstruction found in early childhood.

2. A widened nasal root or unusually large pupillary distance.

3. Presence, simultaneously, of an external encephalomeningocele.

4. A tumor arising high in the nose over which the septal mucous membrane appears to continue.

5. Signs indicating cranial cavity communication, as fluctuation synchronous with the pulse and respiration.

6. Post-operative cerebro-spinal rhinorrhea.

X-rays, in any position, offer little aid in diagnosis despite pathological defects up to one centimeter in diameter which have been demonstrated at post mortem. Diagnostic puncture or biopsy is not recommended, being dangerous despite usual precautions. One must infer, then, that the best aid in diagnosis is to suspect the possibility of such an anomaly.

REPORT OF A CASE

The patient S. M. was seen in the nose and throat clinic of the California Babies' and Children's Hospital at the age of two weeks. The complaint was noisy respiration and difficulty in nursing properly since birth because of apparent nasal obstruction. The child appeared well developed and well fed. A physical examination by the pediatrician had disclosed no pathology other than a sero-mucoid discharge from the left side of the nose, associated with a similar type of post-nasal discharge. Small catheters could be passed readily through each side of the nose into the nasopharynx, eliminating the possibility of choanal atresia. The nasal root appeared abnormally widened; however, in a negro baby this is difficult to be precise about.

Examination of the nose after nasal shrinkage and suction disclosed, in the left side, a bluish-gray, smooth, readily compressible and moveable mass 1 cm in diameter at approximately the level of the inferior turbinate. At first impression it appeared to be a boggy turbinate. The mass, however, did not shrink under decongestants.

The baby was seen at weekly intervals. Though shrinking solutions afforded symptomatic relief from nasal discharge, the mass continued to grow. By five weeks it was fixed at the level of the nasal vestibule completely occluding the left nostril and was no longer compressible. X-ray, digital, and direct examination of the nasopharynx gave no clue as to the origin of the mass.

Suspecting a tumor rather than a herniation a biopsy was done in surgery and a portion of the presenting tip was removed. Very little bleeding was encountered; no rhinorrhea was seen following biopsy. Microscopic examination of the tissue obtained was reported as follows: "The gross specimen consists of two pieces of pinkish-gray, fixed tissue, the larger 6 mm in greatest dimension. Histologic examination shows that the tissue is covered in places by stratified squamous epithelium and in other areas by pseudostratified columnar epithelium. The smaller piece of tissue is of central nervous system type and shows many ganglion cells which lie in a loose fibrous glial stroma. The tumor is infiltrated in places with a few small round cells. In one area there are a number of large brown pigment granules of apparently hematogenous origin. Two round refractile, partially calcific structures are seen in one area. One margin of the tissue is covered by fibrinous debris and leukocytes. There is no demonstrable evidence of malignancy in the material submitted. The diagnosis is: Ganglial neuroma of nasal-pharyngeal area" (Fig. 1).

Consultation was held with the tumor clinic and excision by the rhinologist was suggested. The radiologist felt this tissue was not radiosensitive. Since the

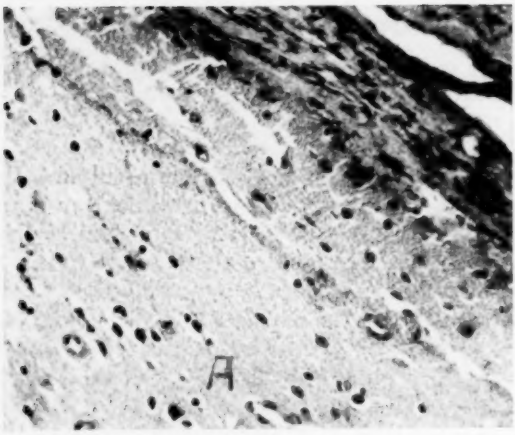

Fig. 1.—Photomicrograph—nasal tumor.

literature on ganglial neuroma indicated extirpation of such tumors was the treatment of choice the infant was prepared and surgery was done.

Our approach was made through a left lateral rhinotomy. It was found necessary to excise the left nasal bone and a projection of the ascending portion of the left maxillary bone. Every effort was made to preserve the periosteum of these excised bones by reflecting them away from the surgical field. The rhinotomy was not completed through the filtrum of the lip as exposure was adequate. On opening into the left nasal cavity the bluish-gray mass was found to fill the entire area as far up as could be seen. Careful probing disclosed it was free ventrally, medially, and inferiorly, but firmly attached laterally in the ethmoid area. Though attached in the ethmoid area it did not appear to originate from it, and separation laterally by blunt dissection was not too difficult. The septal mucous membrane appeared to continue over the tumor. Gradually the belly of the mass was freed progressively upward to the root of the nose.

To our concern the neck of the tumor disappeared through a large dehiscence in the left cribriform plate, measuring approximately 4 x 8 cm. It was decided to ligate and snare the thin pedicle from which the tumor was suspended and this was done. No cerebrospinal fluid leakage from the stump was observed. In an attempt to close off this dehiscence a 2 cm disc of septal mucous membrane was excised, plastered over the opening as a free graft, and packed in place with petrolatum gauze.

The reflected periosteum of the nasal bone was returned to its position and the excised nasal bone reposited. The rhinotomy was closed with fine silk for the subcutaneous tissue and dermoline for the skin, in interrupted sutures. A nasal feeding tube was left in place through the unopened side.

The infant was placed in isolation and maintained on a full complement of antibiotics. That evening the rectal temperature rose to 101.4. Attention was given to proper feedings through the nasal tube, humidification, and hydration. The temperature remained below 101 (rectal) until the evening of the third day when it rose to 104 (rectal). Examination disclosed a markedly inflamed throat and some diffuse redness of both ear drums. No bulging or loss of drum markings

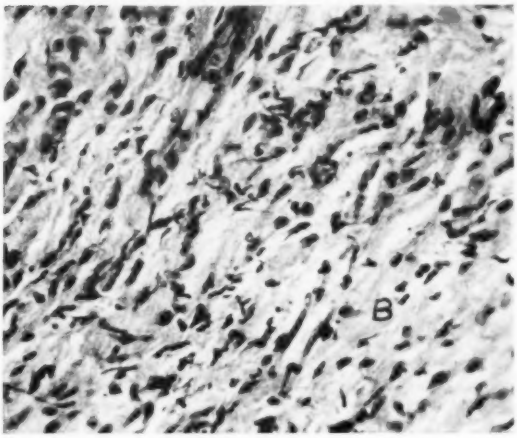

Fig. 2.—Photomicrograph—nasal tumor.

was seen. Nose drops were added to the therapeutic regimen and by the following morning the temperature dropped to 100 (rectal). The mother reported she had visited the baby that day, although she did not feel well. The next day she developed a typical upper respiratory infection. For the next five days the infant did exceptionally well. No signs of meningitis developed and at no time did the child evidence more than a mild annoyance at having its head flexed. On the seventh day the nasal pack was removed; careful search disclosed no evidence of sloughing of the free graft closing the cribriform dehiscence. Recent examination with the nasopharynscope suggests it is still in place.

The pathological report was quite surprising in view of the biopsy report. The report reads: "Specimen: 'Nasopharyngeal tumor.' The gross specimen consists of a piece of soft, glistening, reddish-gray tissue which measures 22 mm in greatest dimension. In addition, there are several bits of soft, glistening, reddish-yellow tissue, the largest measuring 10 mm in greatest dimension.

Histologic examination shows brain tissue of cortical type in which there is evidence of chronic inflammation with hematogenous pigmentation which is apparently secondary to the previous biopsy. There is no evidence of a specific tumor. The findings suggest that this specimen is derived from some variety of congenital anomaly, probably of the character of an encephalocele" (Fig. 2).

It is certain that had we any idea of the true nature of the tumor we would not have approached the problem in this way. The neurosurgical consultant now advises only careful watching of the progress of the baby, especially during episodes of upper respiratory

infection. If the infant develops meningeal complications with such respiratory infections then closure of the cribriform dehiscence through an intracranial approach will be done. The infant has had three such upper respiratory infections since surgery with no untoward sequelae.

SUMMARY

The problem of nasal encephalomeningocele is discussed. The literature is summarized and a case of a patient surviving intranasal extirpation is presented.

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XIII

TOXIC EFFECTS OF DIHYDROSTREPTOMYCIN UPON THE CENTRAL VESTIBULAR MECHANISM OF THE CAT

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Dihydrostreptomycin, the product of the catalytic hydrogenation of streptomycin and possessing the same antibiotic activity as the parent substance, was introduced as an agent in the treatment of chronic diseases, such as tuberculosis, in the hope that it would exhibit a lesser degree of neurotoxicity than streptomycin itself had shown.

Early experimental studies supported the hypothesis that dihydrostreptomycin is less injurious to the vestibular apparatus than is streptomycin. Edison and her co-workers[1] found in cats given equal amounts of streptomycin and dihydrostreptomycin that ataxia and loss of nystagmus appeared later and were less severe with dihydrostreptomycin. Hobson et al,[2] in a small series of cats given both antibiotics, concluded that dihydrostreptomycin produced essentially the same neurotoxic signs as streptomycin, but that possibly these toxic manifestations were delayed when dihydrostreptomycin was employed.

The earlier clinical reports also seemed to indicate that dihydrostreptomycin is less injurious to the vestibular system. Domon

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Presented at the Sectional Meeting of the American College of Surgeons, Atlantic City, New Jersey, February 12, 1952.

This study was aided by a grant from the National Institute of Health of the United States Public Health Service.

and his associates[3] declared dihydrostreptomycin to be much less toxic than streptomycin on the vestibular apparatus after studying two groups of patients which had been receiving similar dosages of the two drugs over a period of 90 days. Hobson and his co-workers found that evidence of vestibular damage appeared later and less uniformly in patients given 5.0 gm of dihydrostreptomycin daily than it had in patients given 3.0 gm of streptomycin daily.

More recent reports, however, indicate that dihydrostreptomycin may cause serious damage to the vestibular mechanism, when given in high daily dosage over prolonged periods of time, and especially when the patient has renal impairment. Carr and his colleagues[4] have described 10 patients receiving dihydrostreptomycin in whom signs of vestibular dysfunction occurred. The impairment in these patients varied from slight hypofunction to complete loss of all vestibular function. These workers believe that even in a patient with normal renal function a dosage of dihydrostreptomycin in excess of 1.0 gm every twelve hours should be avoided.

Perhaps even more disturbing than the recorded instances of vestibular dysfunction are those reports of losses of hearing in patients receiving dihydrostreptomycin. It is recognized that streptomycin occasionally affects cochlear function, but it was never considered a serious drawback to streptomycin therapy since impaired hearing occurs probably in less than one per cent of patients receiving the drug. In the series of 10 patients reported by Carr et al. two developed hearing losses, recorded on serial audiograms, while on dihydrostreptomycin. Bernard, Paley and Arnaud[5] studied 17 patients receiving 3.0 gm of dihydrostreptomycin daily. Of the 17, all but three developed hearing impairment. Five of these patients became completely deaf, three had important hearing losses and six had some degree of hearing deficiency. Shane and Laurie[6] reported on 21 patients with pulmonary tuberculosis who were given 2.0 gm of dihydrostreptomycin daily for 90 days. None of the 21 showed vestibular dysfunction but two of the patients developed severe hearing loss. The hearing impairment occurred on the 89th day of therapy in one patient, and in the other, the loss did not become apparent until 24 days after the drug had been withdrawn. Van Goidsenhoven and Stevens[7] concluded that dihydrostreptomycin is extremely toxic for the cochlear system. These authors declare that the antibiotic should be used for short term therapy only, and they further recommend that the use of dihydrostreptomycin by the intrathecal route be abandoned. Glorig[8] studied 211 patients with tuberculosis receiving 2.0-3.0 gm of dihydrostreptomycin daily for three months or more. He found that 31 per cent of these pa-

tients showed hearing losses of at least 30 decibels. Glorig believes the damage to the hearing is irreversible in all cases in which a significant hearing loss exists.

In previously published papers[9, 10] we have described damage to the central portion of the vestibular apparatus in cats which had been given large toxic doses of streptomycin parenterally. These pathologic changes were located in and about the Purkinje cells of the flocculus and nodulus of the cerebellum, in the dentate and fastigial nuclei of the cerebellum, in the lateral (Deiters') and medial (triangularis) vestibular nuclei of the medulla oblongata, in the ventral cochlear nuclei, and in the reticular substance, the acoustic tubercle, the trapezoid body and among the anterior horn cells of the cervical portion of the spinal cord. These widespread changes, in the main, consisted of pyknosis and shrinkage of the nerve cells. Occasionally, there was actual cell loss.

It, therefore, seemed logical to administer large toxic doses of dihydrostreptomycin to another series of cats to determine whether similar pathologic changes could be demonstrated. It is the purpose of this paper to report on our findings in these animals.

Two methods of investigation were employed: (1) vital staining with trypan blue; and (2) histopathologic examination of brain sections stained with cresyl violet and phosphotungstic acid.

All of the animals used were healthy adult cats with normal vestibular function, as evidenced by the turning test. Inasmuch as the cats used in the dihydrostreptomycin experiments were from the same source as those in the streptomycin studies, the same control animals were employed for both investigations. The controls were kept in the same quarters, fed and prepared in the same manner except that these animals did not receive either streptomycin or dihydrostreptomycin.

VITAL STAINING EXPERIMENTS

Under normal conditions, the injection of certain dyes, such as trypan blue, into the blood stream of an adult animal will result in vital staining of all the tissues and organs of the body except those of the central nervous system. However, when an inflammatory or necrotizing lesion has occurred in the central nervous system, that area will then take up the vital stain. These facts have given rise to the concept of the hematoencephalic barrier. Just where this barrier lies is a matter of dispute. It may be in the endothelium of the capillaries of the brain and spinal cord, or it may reside in the

nerve cells themselves. A discussion of these two hypotheses was presented in one of our previous articles.[9]

Control Studies: Fourteen healthy adult cats were anesthetized with a two per cent solution of nembutal in normal saline administered intravenously. Each animal was then given an injection of 20 cc of sterile isotonic saturated solution of chemically pure trypan blue into the right carotid artery in the direction of the heart. In the cat, when trypan blue is thus injected, a high concentration is carried via the right subclavian and vertebral arteries to the base of the brain. This produces a deep staining of the meninges of the medulla and cerebellum. Forty-five minutes to one hour after the injection of the dye, these cats were sacrificed by an injection of 25 cc of 10 per cent formalin into the left carotid artery in the direction of the brain. The brain and spinal cords were immediately removed and prepared for pathological study.

In none of the control animals was there gross or microscopic evidence of trypan blue staining of the central nervous system tissue except around a stab wound of the cerebral cortex which had been inflicted in several of the controls to test the effectiveness of the trypan blue technic. In eight of the nine controls (89 per cent) in which a stab wound was made, the nervous tissue about the traumatized area showed gross and microscopic trypan blue staining.

Toxicity Studies: In our previous work with 16 cats made toxic by parenterally administered streptomycin in three divided daily doses of 0.33 gm each, intracellular trypan blue was found in eight (50 per cent) of the brain specimens. Twenty additional cats which had received a single daily dose of 1.0 gm of streptomycin failed to exhibit the dye in the brain cells in a single instance.

In the series being reported here, seven healthy adult cats with normal vestibular function each were given 1.0 gm of dihydrostreptomycin subcutaneously in three equally divided doses daily for 14 consecutive days. All of these animals showed ataxia within the first week of administration of the antibiotic. Turning tests were done on the seventh and fourteenth days. By the fourteenth day the vestibular eye responses to turning were absent in every cat.

Each of the seven cats was then injected with trypan blue solution, and sacrificed, in the same manner as described for the controls. The brains and spinal cords were immediately removed and prepared for pathological study. In only one (14.3 per cent) of the brain specimens was there evidence of trypan blue staining. In this specimen, the dye could be seen with the naked eye in the cere-

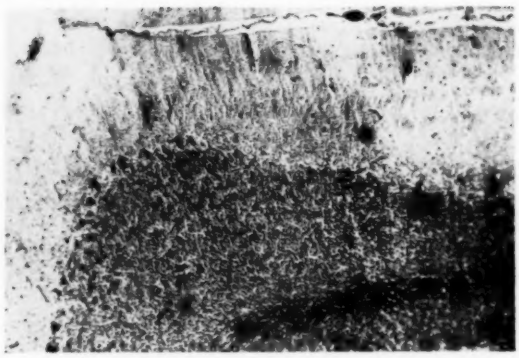

Fig. 1.—Photomicrograph of a single convolution of cerebellum stained with cresyl violet. The Purkinje cells of the righthand portion are normally stained. Those of the left portion are pyknotic, shrunken and partially disintegrated. (x 100)

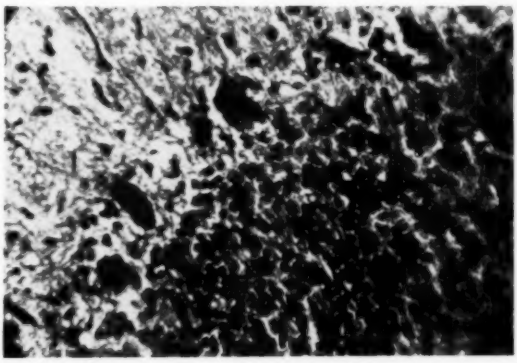

Fig. 2.—Photomicrograph showing a number of Purkinje cells deeply stained with cresyl violet. These cells are from the upper left portion of the cerebellar convolution in Fig. 1. Note the marked degree of structural aberration. (X 430)

bellum and brain stem. Microscopically, the trypan blue granules were present in the nucleus of some brain cells. In still other cells, the dye granules were seen in the cytoplasm as well as in the nucleus. These findings were confined to the Purkinje cells of the cerebellar cortex, most marked in the flocculus, the dentate and fastigial nuclei, the medial vestibular (triangularis) nucleus, and the lateral vestibular (Deiters') nucleus of the medulla oblongata.

ROUTINE HISTOPATHOLOGIC STUDIES

Controls: The brains of 25 cats which had received neither streptomycin nor dihydrostreptomycin were sectioned and stained with cresyl violet and phosphotungstic acid. Fourteen of these animals had been injected with trypan blue previously; 11 animals had not been vitally stained. Of the 25 specimens only one (4 per cent) showed any pathologic findings.

Toxicity Studies: In our streptomycin series of 49 cats definite pathological lesions were demonstrated in 29 (59.2 per cent) of the brain specimens. In 20 of the brains either there was no pathology or the findings were doubtful.

For this study one gram of dihydrostreptomycin was administered daily in three divided doses to each of six healthy adult cats with normal eye responses to the turning test until the vestibular reflexes were abolished. Each animal received 14.0 gm of dihydrostreptomycin over a fourteen day period. The average daily dosage of 1.0 gm per cat was about 0.3 gm per kilogram body weight. This dosage is ten to twelve times the therapeutic dosage of dihydrostreptomycin for an adult man.

These six cats were then sacrificed with an intravenous injection of ether, and the brains and spinal cords were immediately removed for pathological study. After sectioning, these specimens were stained with cresyl violet and phosphotungstic acid. The brains of the seven cats to which dihydrostreptomycin had been administered and then injected with trypan blue were also sectioned and stained with cresyl violet and phosphotungstic acid.

All 13 specimens showed evidence of brain tissue damage. The pathological changes chiefly noted were those of pyknosis and shrinkage of the nerve cells. Occasionally, complete loss of the cell was observed. These changes were particularly marked in and around the Purkinje cells of the flocculus and nodulus of the cerebellum (Figs. 1 and 2). Here could be seen all stages of cell degeneration, varying from pyknosis of the nucleus alone, with or without disso-

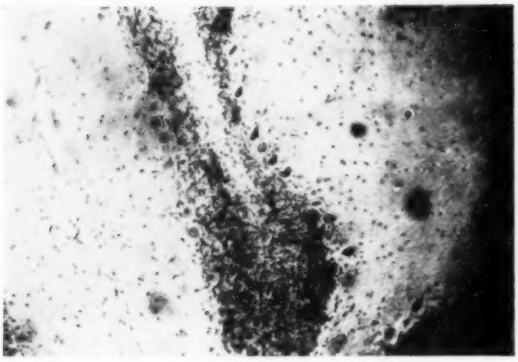

Fig. 3.—Photomicrograph of a single convolution of cerebellum stained with cresyl violet. The Purkinje cells are shown to be affected. Some are darkly stained, pyknotic and shrunken. Others appear quite normal. (X 100).

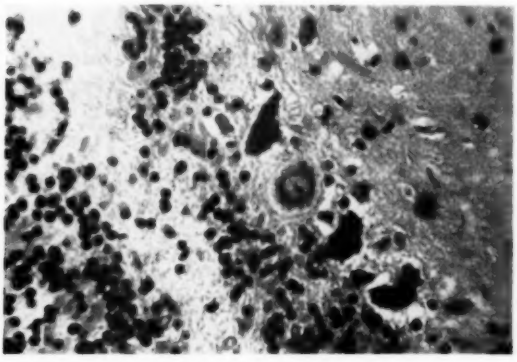

Fig. 4.—Photomicrograph showing under higher magnification the haphazard way in which Purkinje cells were affected in Fig. 3. (X 430)

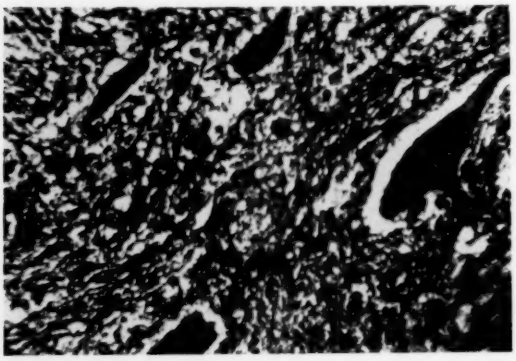

Fig. 5.—Photomicrograph of some pyknotic and shrunken nerve cells in Deiters' nucleus. Note the difference in size, and density of staining, between the abnormal cells on the left of photograph and the normal cell, right. Phosphotungstic acid-hematoxylin. (X 430)

lution of the Nissl substance, to shrinkage and complete loss of the cell. In some areas there was an overlying stag-like dendritic arborization covered with granules. In several of the specimens the Bergmann cells were beginning to develop glial fibres which stained with phosphotungstic acid. Injury to cells in the dentate and fastigial nuclei of the cerebellum was also observed.

Wherever cell damage was found it varied in degree from animal to animal. Furthermore, the toxic effects were manifested in a rather haphazard manner. Perfectly normal cells could be found immediately beside badly damaged cells (Figs. 3 and 4).

Pathological changes less marked than those in the cerebellum were demonstrated in the lateral vestibular (Deiters') nucleus in all of the brains (Fig. 5). In several of the specimens, cell damage was also visible in the ventral cochlear nuclei of the medulla. There were no pathological changes observed in the cerebral cortex in any of the cats.

SUMMARY

Evidence is presented on the damage to the central portion of the vestibular apparatus in cats which had received toxic amounts of dihydrostreptomycin by the parenteral route.

The lesions observed in the central nervous system of these animals were similar in degree and in location to those reported in a previous series of cats to which streptomycin had been administered.

The most marked pathologic changes were present in and about the Purkinje cells of the cerebellum, especially in the flocculus and nodulus. These changes varied from pyknosis of the nucleus to complete loss of the cell. Structural aberrations of the cells of the dentate and fastigial nuclei of the cerebellum were observed.

Less marked but definite damage to the cells in the lateral vestibular (Deiters') nucleus and the ventral cochlear nuclei of the medulla were noted.

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XIV

AURAL VERTIGO

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This survey of cases seen by me since 1945 is reported because my technic of vestibular examination has gained in refinement. It may be possible today to determine objective changes in excitability in patients whose vestibular apparatus has formerly been considered normal and to distinguish between a peripheral and central disturbance of the vestibular apparatus.[2]

One hundred seventy-one unselected cases are reviewed, 46 of the patients developed their dizziness after injuries in military action.

Only those cases are denoted in this paper as true Ménière's disease which show the characteristic triad of a subjective nature: vertigo, tinnitus and deafness. The subjective triad is stressed in contradistinction to the objective symptoms encountered in Ménière's disease to be discussed later in detail.

Classed as Ménière's syndrome are cases in which patients complain of dizziness in attacks, combined only with one of the other symptoms of the mentioned triad. This group has been created, in my opinion, through uncertainty. With some improvement in our diagnostic knowledge the number will decrease considerably. Some of the cases classified today in this group, will prove to be a true Ménière's disease, others will turn out to be due to neurological or vegetative causes. Perhaps a few will remain which we could call vertigo of unknown etiology. The fact that in my statistics the number of patients in this group is only 15 as against 35 of McNally's, supports this prediction.

I listed all older patients with high blood-pressure as arteriosclerotic, if no other reason for the hypertension could be found.

In the toxic-infectious-labyrinthitis group I included also infectious cases of Ménière's syndrome which, according to several authors[3, 4, 5, 6] are today accepted as an isolated form of encephalitis epidemica, affecting exclusively the eighth nerve and caused probably by a virus. It may be the virus of the encephalitis epidemica itself. A quarter of a century ago, I observed with others[3, 4, 5] many such

TABLE I.

DIAGNOSIS AND DISTRIBUTION OF THE CASES.

True Ménière's disease	21
Ménière's syndrome	15
Infectious toxic labyrinthitis	8
Cardiovascular diseases	29
Central nervous system	18
Post-traumatic	46
Funct. vasomotor, neurotic, vegetative	26
Various diseases	8
Total	171

cases, arising in a nearly epidemic form. In the last years their number decreased strikingly. Also this fact proves the infectious nature of this disease which appears from time to time in small epidemics.

To this group belong also cases of aural vertigo which arise, with or without cochlear symptoms, after treatment with streptomycin.

SYMPTOMATOLOGY

Vertigo: A great proportion of the patients complained of vertigo without being able to characterize it. Some admitted the feeling of rotation. Mostly the surroundings seem to rotate; more seldom, the patient himself had the feeling of being rotated. The plane of rotation described is mostly horizontal, but may also be vertical, diagonal or combined.

Another kind of vertigo is described as unsteadiness in walking which appears likewise in sudden attacks. The patient complains of staggering or of a feeling of being drawn to one side. Sometimes he feels the ground giving way under his feet. In other cases he has "blackness before his eyes." This last kind is encountered very often in vasomotor or intestinal disturbances.

Is sudden onset of vertigo characteristic for certain conditions? Sudden onset was present among my civilian patients in 79 cases of 125 (63%). In cases of Ménière's disease sudden onset was present in 90 per cent, in Ménière's syndrome in only 53 per cent, thus nearly as frequently as the average. This confirms the known fact of sudden onset of vertigo in true Ménière's disease.

TABLE II.

KINDS OF VERTIGO.

TYPE	CIVIL CASES	POST-TRAUMATIC CASES	TOTAL
Feeling of rotation	33	9	42 (25%)
Unsteadiness in walking	23	8	31 (18%)
Vertigo after head movements	3	2	5 (3%)
Blackness before the eyes	8	3	11 (6.4%)
Vertigo without specification of its kind	61	19	80 (47%)
Total	128	41	169

Are complaints of rotatory dizziness in Ménière's disease more frequent than in other groups with vertigo? Rotatory dizziness, according to my statistics, is not characteristic of a special origin. Any pathological irritation of the vestibular apparatus may provoke rotatory vertigo.

a. May it be that patients with a spontaneous nystagmus incline more than others to a rotatory vertigo? This cannot be confirmed by my statistics. Among 125 civilian patients I found spontaneous nystagmus in 28 per cent of all cases. Among the cases of rotatory dizziness I found it in only 20 per cent.

b. May it be that patients with spontaneous deviation-reaction of the arms which is a sign of irritation of the Deitero-spinal reflexes, incline more than others to unsteadiness in walking? I found this unsteadiness in approximately 20 per cent of my patients. Spontaneous deviation-reaction of the arms I saw in 61 patients, of which 11, again about 20 per cent, complained of unsteadiness in walking.

c. May it be that patients with spontaneous deviation-reaction of the arms and spontaneous nystagmus incline more than others to rotatory dizziness and unsteadiness in walking? Both spontaneous symptoms were present in 20 per cent (22 times). Four of them complained of rotatory dizziness, nearly the average of 25 per cent. Only three of them (14 per cent) complained of unsteadiness in walking: less than the average of 18 per cent.

There seems to be no causal connection between spontaneous objective vestibular reactions as nystagmus and deviation-reaction of the arms and the subjective phenomena as rotatory dizziness or un-

TABLE III.

TINNITUS: AMONG 171 PATIENTS THERE WERE

Tinnitus present	82 times (47.3%)
Unilateral	35 times (20.3%)
Bilateral	11 times (6.4%)
Not lateralized	36 times (20.3%)
No tinnitus	89 times (52.7%)

steadiness in walking. Perhaps we may conclude from this analysis that the subjective and objective vestibular phenomena proceed by different central pathways.

Deafness: Any kind of deafness may belong to the clinical picture of aural vertigo, not only as internal, but also as middle ear deafness, be it a chronic purulent otitis media or a former suppuration of the middle ear, healed with a dry perforation or a scar of the drum. I found deafness of every kind among 125 civilian patients 79 times (63.2 per cent). The deafness was 54 times (43.2 per cent) unilateral, 25 times (20 per cent) bilateral.

There were 16 pure middle ear affections, 47 pure inner ear lesions and 16 cases of a mixed type. Among the 16 middle ear lesions only one had a true Ménière's disease and only one other had a Ménière's syndrome. This is in accord with the general opinion that we have to deal in Ménière's disease nearly always with inner ear lesions. But we have in my opinion no right to exclude, for this reason, the possibility of a true Ménière's disease in a case of a middle ear affection. Even the latter may cause a locus minoris resistentiae and may lead to a true Ménière's disease.

McNally found tinnitus in approximately 75 per cent of his patients as against 47.3 per cent in my statistics. I am not able to explain this difference.

In one case, after labyrinth destruction by intensive radium treatment, the dizziness ceased but the tinnitus remained unchanged, conforming to the experiences of other authors after eighth nerve cutting in similar cases.

VESTIBULAR APPARATUS

Vertigo, the common complaint of all patients reviewed here, is the subjective irritation-symptom of the vestibular apparatus and analogous to tinnitus, the subjective irritation-symptom of the coch-

lea. At the onset of vertigo and even a few months afterwards, the vestibular apparatus may be objectively, completely normal. The longer vertigo exists, the more often we find objective changes in the vestibular excitability, provided that the damaging cause remains effective in the inner ear, as in most true cases of Ménière's disease. I saw such patients who soon after onset of the dizzy attacks, had a completely normal vestibular apparatus, even with the improved methods of examination which I use now.[7] When examined, however, a few months later, there were already objective changes: hyperexcitability or more important, hypoexcitability of the vestibular apparatus.

In the following Table IV are the findings of the vestibular apparatus.

Normal excitability of the vestibule was found in 63 cases (34 per cent) and pathological findings in 119 cases (66 per cent). Compared with McNally's figures, there is a striking fact: the number of normal vestibules is 34 per cent in my statistics, compared with 70 per cent in McNally's. Although I agree with McNally that the findings in the vestibular apparatus have no definite significance for the diagnosis of Ménière's disease or Ménière's syndrome, it seems to me that cases of longer duration of vertigo (approximately from 6 months up) show more frequently objective changes of the vestibular apparatus than cases with shorter duration of vertigo. This is, in my opinion, of some importance.

Here the fact of the refinement in the methods of vestibular examination may be stressed, as I developed them in the last years.[2] This refinement permits in my opinion a more exact differentiation in the diagnosis than before. Due to these methods we may differentiate today between a peripheral or central origin of the vestibular disturbance. In the histories of patients seen before 1945 who are not included in this review, the remark: "Vestibular apparatus normal" will be found more often than in the last years. With these improved methods we may diagnose slight vestibular disturbances which formerly escaped our attention.

This refinement which takes into account a few seconds in shortening or lenghtening of the so-called latent period in calorization, or a few inches in judging of the deviation-reaction of the arms or of the arm-tonus-reaction, compares with similar refinements in neurology. Every experienced neurologist uses quicker or slower response in certain reflexes and similar signs for a more delicate quantitative diagnostic judgment. In the same manner we also should adjust the vestibular methods (mass-calorization, rotation test), to the

TABLE IV.

OBJECTIVE FINDINGS OF THE VESTIBULAR APPARATUS.

	Hyperexcitability		Hypoexcitability		Unexcitability	
	Unilateral	Bilateral	Unilateral	Bilateral	Unilateral	Bilateral
Peripheral	25	9	24	4	5	2
Central	13	9	14	14		
Total 119	38	18	38	18	5	2

delicate level of other specialities such as ophthalmology and neurology.

I syringe the ear with 20 cc of cold water (18-20° C.) for ten seconds. Then I measure the latency period of the vestibular nystagmus (appearance of its first definite slow component) with a stop watch. The patient wears Bartel's spectacles and looks straight ahead. In the same manner the first appearance of the deviation-reaction and the arm-tonus-reaction of both horizontally extended arms is determined. Intensity and exact duration of all mentioned vestibular reactions has to be noted. Then also warm water syringing is performed (20 cc at 44° C.) and the same phenomena observed and noted as before.[7]

One fact has to be stressed here: For many years I examined every patient (before the experimental stimulation of the vestibular apparatus) to see if there were some spontaneous vestibular symptoms present, for instance as deviation-reaction of the arms or arm-tonus-reaction. We see, not infrequently, such spontaneous phenomena in patients whose vestibular apparatus proves in the following examination to be completely normal. If the spontaneous vestibular symptoms are present on more than one examination, such a vestibular apparatus can not be considered as completely normal; the more so, if the direction of the spontaneous deviation-reaction of the arms is identical with the side of the lowered arm in the arm-tonus-reaction. For instance, if both arms deviate spontaneously to the right and the right arm lowers, we may suspect an irritation of the right vestibular apparatus.

I cannot recapitulate here all I said in former papers about the clinical importance of these spontaneous vestibular symptoms. It

would partly correspond to conclusions drawn once by Bárány from the presence of spontaneous past-pointing to one side, because the deviation-reaction of the arms is[8] the principal cause of the past-pointing reaction itself.

Every patient with the aforementioned spontaneous vestibular phenomena, even with negative findings in the vestibular apparatus, is kept under observation. According to my experience, either there develop at a later date objective changes of the vestibular apparatus, which prove the former spontaneous symptoms as forerunners, or the spontaneous symptoms diasappear with the improvement of the patient's health. Also in this latter case the spontaneous phenomena can be considered as the unique vestibular symptoms present at the first examination.

OTHER SYMPTOMS

a. Headaches, heaviness of the head, a feeling of drunkenness, often described in cases of aural vertigo, were present among 125 civilian patients 27 times (21.6 per cent).

b. In some cases patients fall or even collapse during their dizzy attacks. Of five patients of this kind, one proved to be neuropathic, another had petit mal. Three others lost consciousness during attacks, one had epilepsy.

c. In a small number there was definite evidence of allergy in the history and also in the findings (allergic rhinitis, asthma, bronchial asthma, eczema).

When speaking of the triad symptoms in a true Ménière's disease, it must be clear that this marks only the subjective complaints of the patient, without drawing attention to the objective changes of the vestibular apparatus.

This subjective triad was present among 125 civilian patients 42 times (34.4 per cent), among 46 war-injured 28 times (60.2 per cent), thus among all 171 cases 70 times (41 per cent). Objective disturbance of the vestibular apparatus, in addition to the subjective triad, was present among the 42 patients concerned 25 times (20 per cent) among the 28 war-injured 18 times (39 per cent).

The subjective triad together with objective changes of the vestibular apparatus was found most frequently in cases of a true Ménière's disease, as was to be expected. Second in frequency were neurological cases.

In some cases of Ménière's disease the tinnitus was absent. It may be very weak and escape the attention of the patient. Frequently

TABLE V.

CONNECTION BETWEEN DURATION OF VERTIGO AND OBJECTIVE CHANGES OF THE VESTIBULAR APPARATUS (106 CASES).

(A) Duration of vertigo in cases of a normal vestibular apparatus			(B) Duration of vertigo in cases of objective disturbances of the vestibular apparatus	
Up to 14 days	6 patients		13 patients	
Up to 1 month	5 patients	21 (66%)	2 patients	32 (44%)
Up to 6 months	10 patients		17 patients	
Up to 1 year	1 patient		7 patients	
More than 1 year	10 patients	11 (34%)	35 patients	42 (56%)
Total		32		74

we may draw the attention of the patient to this symptom by questioning or he will pay attention to it in the quiet of the night.

It is interesting to analyze the sequence in the development of the various triad-symptoms: tinnitus, deafness and vertigo. Looking over 55 cases of the 5 main-groups (Ménière's disease 20, Ménière's syndrome 15, arteriosclerosis 7, neurological diseases 6, vegetative, neurotic, vasomotor patients 7) we come to the following conclusions:

(1) In cases of a true Ménière's disease all three phenomena may appear in the same frequency as the first symptom. This confirms the known fact that in Ménière's disease cochlear symptoms (tinnitus and deafness) may mark the onset of the disease as frequently as vertigo.

(2) In cases of Ménière's syndrome vertigo is by far the first and most frequent symptom. Because in this group the cochlea is less frequently affected, development of early deafness, especially of the perceptive kind, should evoke the suspicion of a slowly starting true Ménière's disease.

(3) The review of those 55 cases shows vertigo as the most frequent symptom of the triad.

One may assume that vertigo represents in the beginning of its development only an irritation of the vestibular apparatus which in such "fresh" cases may be itself, objectively, completely normal. If, however, vertigo and the basic damaging factor which causes Ménières'

disease, are present a longer time, we may find also objective changes of the vestibular apparatus.

Among the patients without objective changes of the vestibular apparatus, vertigo existed in 66 per cent not longer than half a year at the most, in the greater part of them only one month. On the other hand, among cases of vertigo with objective changes of the vestibular apparatus, only 44 per cent of them noticed their dizziness less than half a year ago. The majority (56 per cent) had dizziness much longer and nearly half of them (35 cases of 74) more than one whole year. This may perhaps allow the cautious conclusion that the presence of vertigo means in the beginning only a state of irritation of the vestibular apparatus without any objective damage. However should the damaging factor remain for a longer time, it may produce objective damage, perceptible as changes of the vestibular excitability: hyperexcitability or hypoexcitability.

The correctness of this assumption was tested in still another way. The 125 civilian cases were divided into two extreme groups: those who had dizziness at least two years (group A) and those who had it not more than two weeks (group B) to see whether or not the number of objective changes of the vestibular apparatus was the same. For comparison it may be stated that in all 125 cases the number of objective changes was 87 (70 per cent).

Among 32 cases of group A 29 patients (90 per cent) showed objective changes of the vestibular excitability. Among 15 cases of group B only 8 (53 per cent) had objective vestibular changes, thus much less than the average of 70 per cent. Because for this comparison only two extreme groups were chosen, only 47 of the 125 cases could be used.

Subdividing those 47 cases according to their diagnosis:

Group A: 1. True Ménière's disease (9 patients). All of these had objective changes of the vestibular apparatus, especially in the form of hypoexcitability or complete unexcitability. The percentage of objective changes was here 100 per cent, even more than the average of 90 per cent for the whole group A.

2. In Ménière's syndrome there were 75 per cent of positive objective vestibular findings, a little more than the average of 70 per cent.

Group B: The percentage of objective vestibular lesions was under the average of 70 per cent.

In spite of the small numbers my statistics seem to confirm the assumption that vertigo of longer duration may lead more frequently

to objective changes of the vestibular apparatus than "fresh" cases of vertigo.

I know very well that my results must be checked in a critical manner on a much larger number of cases. Should it prove true, it may have also some practical consequences. As long as in a case of dizzy attacks we do not find pathological changes in the vestibular apparatus, we may consider the case in the beginning stage and adjust our therapeutical procedure accordingly. In other words, as long as the vestibular findings are negative, the prospects of conservative treatment are better.

To speak, in a true Ménière's disease, of a triad of symptoms, as is generally done, is right, only from a practical point of view. The objective pathological-anatomical facts are somewhat different. The factor causing the Ménière's disease—which shall not be discussed here—influences, first of all, probably the peripheral sensorial apparatus of the labyrinth in both its branches, cochlear and vestibular, and produces here two kinds of symptoms: subjective, in the cochlea as tinnitus and in the vestibule as vertigo,* and objective in the cochlea as deafness, especially of the perceptive type, and in the vestibular apparatus changes in excitability, chiefly hypoexcitability.

The development of Ménière's disease shows many variants: Sometimes we find after only one or two serious attacks of vertigo a fully developed clinical picture of true Ménière's disease, with all subjective and objective symptoms. But this is not the rule. In the majority of the cases we may observe after the first dizzy attacks only the subjective triad of symptoms, the vestibular apparatus at this time being still completely normal. The development of the subjective and objective vestibular lesion normally takes some time and proceeds in stages, in an analogous manner as the development of the cochlear lesion. In Table VI I tried to define these stages more exactly:

Table VI represents, more or less theoretically, the blueprint of the development of a true Ménière's disease. There are many exceptions to this rule:

Stage I will be observed not too rarely, but can never with certainty be diagnosed as the first stage of a starting true Ménière's dis-

*This view of the development of vertigo requires perhaps some modification in the light of newer experiences: disappearance of vertigo in Ménière's disease after S. Rosen's chordectomy. It may be that vertigo develops not in the peripheral apparatus but, through the connection of the chorda tympani, in the vegetative centres. Or vertigo may be caused in the vestibulum but be strongly influenced, again through the connections of the chorda tympani, by the vegetative centres.

TABLE VI.

DEVELOPMENT OF STAGES IN A TRUE MÉNIÈRE'S DISEASE.

STAGE	VESTIBULAR APPARATUS		COCHLEAR APPARATUS		DURATION OF VERTIGO	REMARKS
	SUBJECTIVE VERTIGO	OBJECTIVE LESION	SUBJECTIVE TINNITUS	OBJECTIVE INNER EAR LESION		
I	+	O	+	O	Few weeks or months	Start of the Ménière's disease
IIa	+	+	+	O	Generally more than 6 months	May be interchanged with stage IIb
IIb	+	O	+	+	Same time	Subjective triad-true Ménière's disease
III	+	+	+	+	Approximately 1 year	Fully developed true Ménière's disease
IVa	+	+	+ or O	Nearly complete deafness	After 1-2 years or more	Advanced case
IVb	+ or O	no response	+	Inner ear lesion as in III	Same time	May be interchanged with IVa
V	mostly O	no response	+ or O	Complete deafness	After years	Final stage

ease. Every experienced otologist in such cases will have the suspicion of Ménière's disease, but will hardly be able to diagnose it.

Stage IIa and IIb as well as stages IVa and IVb may be interchanged, according to which symptoms, the cochlear or vestibular ones, developed first. The real happening depends on factors hitherto not known to us.

It is well-known that the true Ménière's disease may stop in its development at every stage. It may even improve, due to some adequate treatment, but also without any treatment at all, again for reasons not known to us.

In the final stage V there will be total or nearly total deafness with or without tinnitus and unexcitability of the vestibular apparatus without vertigo. This final stage is no theoretical construction, because I have seen such a case.

CONCLUSIONS

Although the primary damage in Ménière's disease arises probably, according to Hallpike and his co-workers, in the peripheral organ, this peripheral affection may influence in every phase of this long-lasting process the cochlear and vestibular centres. We know this influence on the centre in cases of tinnitus, with the exactitude of an experiment: after cutting of the eighth nerve or destruction of the peripheral organ to combat dizzy attacks, vertigo may disappear, but tinnitus often remains. From this happening also McNally concludes a central origin of the tinnitus and says: "If that is the case, then there must be multiple lesions and the labyrinthine lesion is merely one part of the pathological process responsible for the syndrome of aural vertigo."

I would not go so far as McNally and assume multiple processes. It may be that the original peripherally caused cochlear irritation proceeds to the central hearing sphere, producing there a functional irritation, experienced by the patient as tinnitus. This central irritation may persist, even after destruction of the peripheral organ. Perhaps the phenomenon known as "phantom-pain" in an amputated limb may show similar relations.

The happening in the central vestibular sphere could be explained in a similar manner. A few years ago, I advanced the thesis of physiological central mechanisms of checking, the function of which is to suppress the vestibular reactions arising after experimental or spontaneous vestibular stimulation. This suppression is very important, because the vestibular reactions (nystagmus Deitero-spinal reflexes as deviation-reaction, and so on) endanger our vital equili-

brium. I assumed similar mechanisms of checking also for the subjective vestibular reactions as for instance vertigo. If there were no such mechanisms of checking for the vertigo, dizziness would arise at every movement of the head and make our daily life impossible.[9]

We may, perhaps, assume in an analogous manner similar central mechanisms of checking also for the cochlear apparatus. There exist already certain devices in the peripheral organ, destined to keep away from the cochlea extremely loud sounds. Such devices are for example fixation of the drum and stapes through muscle action, the balance between both fenestrae and others. There may be also a mechanism of checking in the central hearing sphere to damp excessive sound-irritation for safety reasons. This may also be valid for tinnitus. There may be always, also in normal ears, tinnitus or more or less strong head-noises which are not experienced because of the action of those central physiological mechanism of checking the sound. If, however, one or more of these mechanisms is eliminated or partly out of action, due to some disease, tinnitus may occur. The elimination of the peripheral organ is then unable to stop the tinnitus.

SUMMARY

In 171 cases of aural vertigo, 125 of them from the civilian practice, 46 war-injured, vertigo, tinnitus, deafness and vestibular disturbance are discussed in detail.

(1) Kind of vertigo: Twenty-five per cent of the patients suffer from rotatory vertigo, 18 per cent complain of feeling of unsteadiness in walking. The remainder complains of vertigo in general terms, of blackness before the eyes and the like.

(2) Deafness has been found in 115 cases (67.6 per cent).

(3) Eighty-one patients complained of tinnitus (47.2 per cent).

(4) There was hyper- or hypoexcitability of the vestibular apparatus, in 119 cases (70 per cent).

(5) The triad: vertigo, tinnitus and deafness were present in 70 cases (41 per cent).

(6) Triad-symptoms with objective changes of the vestibular apparatus were observed in 24 per cent.

(7) Based on these statistics we may assume that vertigo of the vestibular apparatus without objejctive changes is in the majority of cases not older than six months. On the other hand, objective lesions of the vestibular apparatus will more often be found in cases

of vertigo of longer duration than half a year. Therefore, vertigo may be considered as a symptom of irritation of the vestibular sphere which may lead to objective lesions of the vestibular apparatus, if the basic damaging cause exerts its influence for a longer time.

In consequence of this assumption a division of true Ménière's disease in five well characterized stages has been advanced. In this division the presence or missing of objective lesions of the vestibular apparatus have a special significance, also in the therapeutic direction.

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XV

CARDIOSPASM ASSOCIATED WITH PNEUMONITIS, BRONCHIAL GRANULOMA AND BRONCHOLITHIASIS

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In advanced cases of cardiospasm the elongated and dilated esophagus may contain so much retained fluid and food that spilling over into the larynx, trachea, and bronchi frequently occurs. This is most common during sleep when the protective reflexes are less active. It has also been demonstrated during waking hours, especially in debilitated individuals. This periodic regurgitation may result in the development of chronic bronchitis and a variety of pulmonary diseases ranging from lung abscess to fibrosis.

The simultaneous existence of aspiration pneumonitis and bronchial granuloma with broncholithiasis in a case of cardiospasm, appears to be an unusual finding worthy of recording.

REPORT OF A CASE

H. G., aged 73, presented himself on May 10, 1948, with chief complaint of frequent and annoying clearing of the throat because of accumulations of large amounts of mucus. This had been present for over a year. He had no dysphagia, no unusual shortness of breath, nor stomach complaints. He had always been in good health until recently. Six months previously another physician had hospitalized him for pneumonia in the right lung.

On examination the nose, sinuses, and pharynx showed no unusual changes. Mirror laryngoscopy revealed a large amount of retained secretions in both pyriform sinuses. During the examination some of the stringy material was seen spilling over into the larynx, causing the patient to clear his throat. There was no evident palatal or pharyngeal paralysis. An obstructive lesion in the esophagus was presumed to be present.

The report of the x-ray examination of the esophagus and chest, performed on May 10, 1948, was as follows:

From the University of Colorado Medical Center.

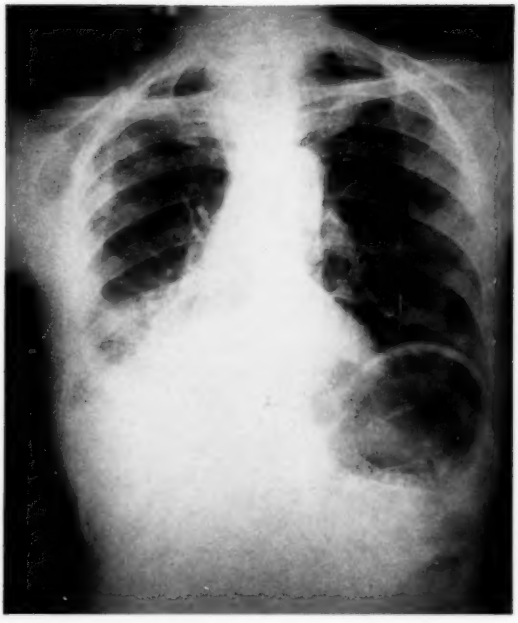

Fig. 1.—Film of chest showing pneumonitis of right lower lung field with obscuration of right hemidiaphragm.

"Fluoroscopic examination shows a large gas bubble in the fundus of the stomach with moderate elevation of the left hemidiaphragm. There is diffuse obscuration involving the lower portion of the right lung field, with little or no diaphragmatic movement on this side. Barium given orally, shows the esophagus to be widened and elongated, but demonstrates no definite obstruction at the cardia. Serial films show evidence of marked esophageal dilatation with smooth narrowing at the lower end and with definite tortuosity due to elongation. In the Trendelenburg position there is no evidence of hiatus hernia. The lateral study of the cervical region following oral administration of barium rules out the possibility of Zenker's diverticulum, but there is considerable retention of barium in the region of the larynx.

A single postero-anterior projection of the chest shows evidence of extensive pneumonitis involving the right lower lobe with obscuration of the right hemi-diaphram, apparently due to some fluid in the pleural cavity on this side. Impression: Achalasia of the cardia

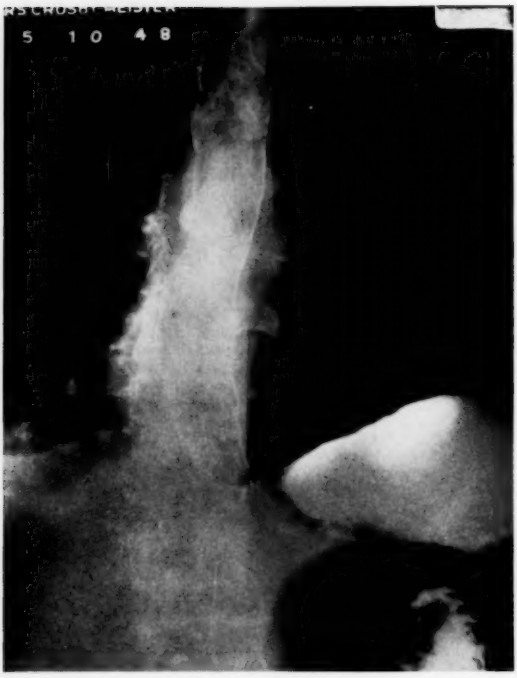

Fig. 2.—Film showing esophageal dilatation with rather smooth narrowing at the lower end, and tortuosity. The two broncholiths may be seen to the right of the mid-esophagus.

with esophageal dilatation and elongation; aspiration pneumonitis, right base."

The patient was again questioned regarding dysphagia, regurgitation of undigested food, substernal distress, or vomiting, but he made light of these symptoms. The size of the dilated esophagus as seen on x-ray would indicate the existence of cardiospasm over a number of years. There had been no appreciable loss of weight.

Esophagoscopy and bronchoscopy were performed on May 14, 1948.

The esophagus contained a large amount of fluid and undigested food particles. The walls of the esophagus appeared greyish, pasty, and smoother than usual. The esophagoscope passed through the hiatus fairly easily. No ulceration or tumor masses were encountered.

Fig. 3.—Film showing improvement in the infiltration in the right lung base (3 months after bronchoscopic removal of the granuloma, and broncholiths, as well as hydrostatic dilatation of the esophagus.

At bronchoscopy a moderate amount of mucopurulent material was aspirated from the trachea and right bronchial tree. A granulomatous mass about 8 mm in diameter was found on the medial wall of the right main bronchus 1½ cm below the carina. Several pieces were removed from the granuloma with bronchoscopic biopsy forceps, resulting in rather free bleeding. Bronchogenic carcinoma was considered.

After bronchoscopy, when the removed tissue was palpated, two hard calcareous spiculated masses, each about 3 mm in diameter, were found incorporated in the biopsy specimens.

The pathologic report follows: "The tissue specimen is composed of a few minute soft tissue fragments and two small, irregular shaped calcareous masses, one measuring 3, the other one 3½ mm in diameter.

Microscopic examination of the soft tissue specimens reveal an inflammatory granuloma. The inflammatory exudate consists of

polymorphonuclear cells, lymphocytes, plasma cells, and lesser numbers of fibroblast cells. The vascularity is considerably increased throughout the entire sectioned area, and there are scattered ecchymoses and blood pigment deposits.

Diagnosis: Inflammatory granuloma of bronchial wall."

One week later two esophageal dilatations were carried out with Plummer's hydrostatic dilators, three days apart, using water pressure up to six pounds.

Subjective and objective improvement in the patient's condition was soon noted, as the spilling over into the larynx abated. He gained ten pounds in a period of three months.

Esophagoscopy and bronchoscopy were repeated on August 19, 1948. The esophagus contained only a small amount of retained fluid. The granulomatous mass in the right main bronchus was half its previous size. It was again removed and its base cauterized with 10% silver nitrate.

A check x-ray of the chest and esophagus on August 24, 1948, was reported as follows: "There is marked improvement in the previously described infiltration in the right base. There is still obliteration of the costophrenic sulcus on this side, probably from thickened pleura. The left lung field remains clear. Following oral barium the esophagus still shows evidence of cardiospasm but there has been marked decrease in the size of the esophagus since the last examination. There is only very slight obstruction at the esophageal hiatus."

The patient was last seen on March 10, 1952, and still appeared to be in good health.

COMMENT

This case illustrates that it is easy to overlook both the pneumonic involvement and the cardiospasm with which it is associated. The symptoms referable to the cardiospasm itself may either not exist or be subordinate to the respiratory symptoms. Studies have shown that in almost 40% of such cases, the initial complaints were referable to the pulmonary complication rather than to the esophageal disease.[1] The finding of accumulated secretion in the pyriform sinuses prompted barium study of the esophagus, otherwise the existing pathology might have remained undiagnosed in this patient.

The association of cardiospasm and pulmonary disease has not been too frequently emphasized as evidenced by the fact that up to 1951 only 46 such case reports had been published. Breakey, Dotter, and Steinberg[1] added 17 to this number, raising the total to 63. The relative frequency of the pulmonary complications encountered were: 1) fibrotic or nonspecific infiltrations 44.8%; 2) lung abscess 12.5%; 3) pneumonia 12.5%; 4) pulmonary tuberculosis 7.8%; 5) bronchiectasis 4.8%; 6) oil-aspiration pneumonia 4.8%; 7) pleural effusion, asphyxia due to massive aspiration and pneumothorax 6.4%. In over half of the cases the right lung alone was involved and in almost an additional third, both lungs were affected.

The pneumonic lesions are generally of the chronic type with tendency to repeated exacerbations, due to recurring insults by the aspirated material. In a second type the pneumonitis may assume an acute character with long periods of intervening relatively good health. Occasionally a patient may die suddenly of massive aspiration of esophageal contents.

Recognition and treatment of the existing cardiospasm is of course urgent. Whether the treatment should be by hydrostatic dilatations or by surgical methods need not be discussed here. Many cases of cardiospasm with pulmonary complications have been controlled by esophageal dilatations. I recently performed a bronchoscopy on a sixty-two year old female with cough of more than five months duration in whom an infiltrative process in the right lower lung field suggested carcinoma. Following bronchoscopy and esophagoscopy, cardiospasm and the associated aspiration pneumonitis were established. Hydrostatic dilatation of the esophagus was carried out and the pulmonary symptoms cleared.

The presence of pneumonitis together with broncholiths and an infectious granuloma in the bronchus of the patient herein presented lend added interest. Evidently the aspirated material not only produced pneumonitis of the lung base, but also, by irritation, a chronic inflammatory process in the bronchial wall as well as calcareous concretions.

Broncholiths when coughed up or removed bronchoscopically give rise to a condition known as broncholithiasis. Until 1941, according to Zahn,[2] only 26 cases of broncholithiasis had been reported in the English literature. He mentioned a total of 71 cases in the literature up to 1946. About half of these were diagnosed by expectoration of the calculi. I recently encountered a patient who had coughed up his seventh broncholith. He had been sent to Den-

ver on account of hilar calcifications. One patient was recently reported to have coughed up 103 stones.[3] Broncholiths have been described varying in size from granules to 139 gr.[3] They occur as a rule in adults; over two thirds in the age group ranging between 40 to 59 years.

In the opinion of most authors broncholiths usually originate as a result of calcification of peribronchial lymph nodes in pulmonary infections. The frequency of hilar lymph node involvement in primary tuberculosis of childhood with its not uncommon tendency to perforate the bronchial wall, thus causing the caseating nodes to be expelled early in the course of the disease, has recently been stressed.[4] When the tuberculous glands are not thus elminated, continuous accumulations of calcium deposits result in the formation of one or more stony concretions which may become extruded in adult life in the form of broncholiths. Under these conditions tubercle bacilli have been found in only a surprisingly small percentage of the reported cases.

Schmidt, Claggett and McDonald[3] mentioned that broncholiths may also originate in the lumen of the bronchus. Thus aspirated foreign bodies and endobronchial secretions may act as nuclei for deposition of calcium. Jackson and Jackson[5] differentiated bronchial concretions into: 1—pneumoliths (cast off calcified caseous masses ulcerating through the bronchial wall); 2—broncholiths (formed by deposit of lime and magnesium salts where pathologic secretions have accumulated in chronic bronchial disease, usually bronchiectasis); 3—silaceous broncholiths (small particles of irregular cylindric shape probably formed in the small bronchi). Curiously, broncholithiasis has rarely, if ever, been found in a patient with silicosis. In the 41 cases reported in the Mayo Series there was no mention of any caused by aspirated secretions secondary to esophageal disease. I have found no record of this occurrence in any of the other articles I have reviewed.

The symptoms of broncholithiasis are not sufficiently characteristic to readily differentiate it from the pulmonary diseases. Cough, hemoptysis, and the signs of bronchial obstruction are the most common and frequently have led to exploratory thoracotomy. Neither physical findings nor roentgenologic evidence are particularly diagnostic. Only 3 of the 41 cases in the Mayo Series were discovered by x-ray. It is interesting that in 10 of their cases the involved bronchus was found strictured or obliterated by granulation tissue, thus raising the possibility of endobronchial tumor.

They stressed that the bronchial mucosa usually returned to normal after the broncholith was removed.

The x-ray cannot be depended upon to delineate the broncholiths and they usually have been confounded with calcified nodes. After reviewing the films of my patient, the broncholiths could be identified in an over-exposed film taken to bring out the dilated barium-filled esophagus (Fig. 2).

The possibility that the broncholiths could have originated extra-bronchially should be considered, yet it would hardly be likely in view of the negative history of previous pulmonary disease. Since the pneumonitis, granuloma formation, and the broncholiths coexisted, it is fair to assume that they derived from a common source. This was provided by the long continued irritation of the aspirated fluids, secondary to the cardiospasm, which was undoubtedly the exciting factor in this multiple pulmonary pathology.

CONCLUSIONS

A case of cardiospasm or achalasia of the esophagus associated with aspiration pneumonitis, bronchial granuloma, and broncholithiasis is presented.

The combination of cardiospasm and aspiration pneumonitis is a well recognized, though frequently overlooked, entity.

Their association with bronchial granuloma and bronchiolithiasis appears to be rare and worth reporting.

Restoration of the patient's health by hydrostatic dilatation of the esophagus and successful bronchoscopic treatment of the disease in the bronchus and lung are recorded.

227 16TH STREET.

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XVI

SELECTION OF THE OPERATION FOR CANCER OF THE LARYNX

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The significant factors, in considering the type of operation to be selected in the treatment of laryngeal cancer, are relatively few. An intimate knowledge concerning their validity, however, is of utmost importance, for depending on such a selection, a human life is held in the balance.

First, and of basic import, is the fact that untreated cancer is a fatal disease. That means other than surgery are employed, and successfully so, in the treatment of cancer, is not a matter of dispute; the pertinent fact in the consideration of laryngeal cancer is that surgery is employed successfully more often than is any other form of treatment. Of lesser importance is the consideration given to the preservation of laryngeal structure or function. This consideration must be distinguished from sympathy for the patient, for such emotional influences constitute one of the cardinal reasons leading to local recurrence in cancer of the larynx.

It is not always a simple matter to obtain a full appreciation of the extent of a malignant lesion of the larynx. In spite of the fact that this structure is readily accessible to examination both by mirror and by direct means, the determination of submucosal invasion, or of pre-epiglottic or subglottic extension, is often, clinically, an impossibility. One must rely on the axiom of adding half again to the visible tumor in order to obtain a real estimate of its actual size.

After a diagnosis of cancer is made, and one is aware of all the physical characteristics pertaining to this patient and his tumor, a consideration of the following criteria must be entertained in order to select the proper surgical method:

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Read before the Philadelphia Laryngological Society, February 3, 1953.

1. Location and Extent of Growth.
2. Presence or Absence of Impairment of Motility.
3. Presence or Absence of Metastases—Local or Remote.
4. Grading According to Broders' Classification.
5. General Condition of the Patient.

Although cordal lesions are so situated as to be productive of voice change, and so are apt to be diagnosed early in their development of voice change, and so are apt to be diagnosed early in their development, it is better that they be referred to according to their apparent extent, and to their location or site in the larynx, rather than that they be classified as "early." Thus, "a small ulcerated granulomatous mass limited to the middle third of the left cord" is more descriptive than "an early lesion of the left cord."

Carcinoma entirely contained on the vocal cord, which cord is free of impairment of motility, is the type of lesion properly selected for thyrotomy. In electing to perform thyrotomy for carcinoma, however, it is well to eliminate the thought of conservation from one's mind; it is reiterated that one is dealing with a deadly disease, and complete resection of the lesion must be of first consideration. And although reports of good results from partial cordectomy are contained in the literature, the same reports indicate that incomplete removal is the principal cause of local recurrence.

The following salient statistics will substantiate these thoughts. In reviewing 193 cases of cancer of the larynx operated on by the technic of thyrotomy, that could be traced for a period of five or more years after operation, it is noted that there are 117 living and free of recurrence, or 73.5 percent. Of this group, there are 42 instances of recurrence, of which 29 occurred locally in the larynx, 6 in regional lymph nodes and 7 as remote metastases. Of the 29 patients with local recurrence, 23 were then operated on by means of laryngectomy and 6 were treated by irradiation. All of the irradiated patients died. And of the 23 operated on by laryngectomy, 12 ultimately died of cancer, and 11 are still living 5 or more years after their laryngectomy. Of the group of 6 with metastases to regional lymph nodes, a radical neck dissection was performed in 4: one is living and well, five years after this operation; all of the others are dead of cancer.

Concerning the 29 patients with local laryngeal recurrence, it is noted in the records that in 7 patients there was evidence of subglottic extension, and in 18 there was extension to either the anterior commissure or to the opposite vocal cord. In the remaining 4, al-

though the carcinoma was limited to one cord, at operation it was seen that there was lateral extension of the cancer, indicating that the lesion was larger than had been anticipated on the basis of the preoperative laryngoscopic examination.

Unquestionably, all of these cases of local recurrence were due to incomplete removal at the time of primary thyrotomy. Therefore, it is evident that laryngectomy should have been the primary surgical procedure. Following this review, then, it is well to further qualify the definition of lesions suitable for thyrotomy: carcinoma, so situated as to be entirely contained on the anterior or the middle third of the vocal cord, without involvement of the anterior commissure or extension to the opposite cord, without subglottic extension or extension into the ventricle, and without any evidence of impairment of motility, is a suitable lesion for thyrotomy. Certain liberties may be taken, at times, by some operators in extending their technic to include lesions that do involve the anterior commissure. The taking of such liberties, however, will be seen to be accompanied by a higher local recurrence rate. The analysis of these cases of thyrotomy likewise shows that to hold laryngectomy as a secondary or reserve procedure in event of recurrence following thyrotomy, is not a justifiable one. The fate of those patients with metastases to regional nodes, or to remote organs, too often was determined long before attention was directed to these metastases.

Conversely, then, and as based on the definition used in the selection of patients for thyrotomy, laryngectomy should be resorted to as the primary operation in instances wherein the lesion extends beyond the confines of the functioning vocal cord, but wherein there is no evidence of either regional or remote metastases. Of 176 patients who could be traced five or more years after operation, treated by laryngectomy there are 81 living and free of recurrence or 58.7 percent. Of this same group, there were 57 instances of recurrence; 41 to regional lymph nodes, 16 to other organs. Of the 41 cases of metastases to regional lymph nodes, radical block dissection of the neck was performed in 21 cases, and of this group, 10 patients died because of cancer; 11 are alive and well 5 or more years after operation. Of the 20 patients not treated surgically, 16 were irradiated: 15 of these died from cancer; 1 is living and well, for a period of 14 years. Of the completely untreated patients with metastases, 8 are still living five or more years after operation; four are living less than five years after operation.

Finally, there is a third group of patients consisting of those who, at the time of initial examination, present signs of local regional

metastasis along with their laryngeal lesion. In such cases, the primary operation is a one-stage total laryngectomy and radical neck dissection. The compilation of statistics pertaining to this group so operated upon is as yet incomplete, principally because of an inadequate postoperative period. It is warranted, however, based on the data available to date, to assert that such a technical procedure is a well-founded one and that additional lives will be saved by incorporating it, along with thyrotomy and laryngectomy, as one of the primary procedures to be considered in the surgical treatment of cancer of the larynx.

Obviously, there is much more to be said concerning the selection of operations. More extensive lesions demand more radical surgery; extrinsic lesions, principally those involving the pyriform sinuses or extending onto the lateral pharyngeal wall, constitute specific surgical problems and are not a consideration in the evaluation of cordal lesions defined in this paper. The facts have been presented with but one thought in mind: to show that in the selection of operations for cancer of the larynx, it is better that one lean toward the radical than toward the conservative side. By holding hard and fast to the definition herein presented, for cases that are acceptable for thyrotomy, would greatly increase the cure rate obtained by this type of operation and would, at the same time allow for a better selection of patients for primary laryngectomy. In so doing more lives will be saved, even though an occasional larynx may be sacrificed unnecessarily.

255 SOUTH 17TH STREET.

XVII

RELIABILITY OF THE EAR-CHOICE TECHNIQUE IN THRESHOLD AUDIOMETRY

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The ear-choice technique as an experimental method for obtaining threshold responses in pure frequency audiometry has been previously described.[1] Briefly, a sample of 20 male, normal hearing, college students was tested by both the Standard Audiometric Technique[2] and the experimental Ear-Choice Technique. The results of this study showed that 98% of the thresholds differed by five db or less as measured by the two techniques, and no difference was greater than 10 db. Product moment r's between the two techniques (computed for each of the six test frequencies) ranged from +0.64 to +0.99. An analysis of variance was used to compare the results of the two tests and no statistically significant differences were found between the thresholds obtained by the two methods.

The subjects used in the preliminary investigation and for whom the previous statistical computations were carried out were all essentially normal hearing individuals. As a further step in the validation of this experimental technique it was deemed valuable to compare the two techniques with a group of individuals known to have serious hearing losses.

The subjects in this study were 20 adolescents enrolled in hearing therapy projects at the University of Illinois. Their ages ranged from 13 to 18 years. The only qualification for the subjects was that their hearing could be measured within the intensity range of the audiometer. No effort was made to select either boys or girls or to designate the type of hearing loss. The subjects used represent the first twenty individuals with hearing loss on whom complete audiograms could be obtained. The mean hearing loss for both ears, six frequencies, as measured by the Standard Audiometric Technique was 43.8 db.

The Microtone, A.D.C. portable audiometer, Model 53C, was set up in a sound-treated room. This instrument was chosen in order

From the University of Illinois.

to utilize the switching feature for changing the stimulus from one ear-phone to the other; any audiometer with a similar feature could have been used in the study. The stimulus tone was "off" until presented by depressing the interrupter switch.

Each of the 20 subjects was tested to obtain a discrete frequency threshold audiogram by both Ear-choice Technique and the Standard Technique. The order in which the two individual tests were administered as well as the choice of ear to be tested first was varied by the use of a table of random numbers. After the headphones were once placed on the subject's head, both tests were administered without removing the headphones.

STANDARD AUDIOMETRIC TECHNIQUE

This procedure represented an attempt to provide a technique which closely approximated the psychophysical method of limits but which could be administered under clinical conditions. The precise steps as used with this technique have been set forth in the preliminary investigation.[3]

EAR-CHOICE TECHNIQUE

1. The subject was asked to respond when he heard the stimulus by touching the earphone from whence it came.

2. The frequencies were tested in the following order: 1024, 2048, 4096, 8192, 512 and 256 cycles per second.

3. Threshold finding procedure:

a. The initial stimulus of 1024 cycles at a sensation level of 30 db was presented first to one ear and then to the other. Tones were presented with sufficient repetitions to assure that no direct pattern of alternation from left to right or right to left was evidenced.

b. If the sound was audible, the frequency dial was then shifted to 2048 cycles. With the attenuator dial remaining constant, the stimulus was presented alternately, at random, to the ears of the subject.

c. With the attenuator dial constant, the remaining frequencies were presented in the same manner.

d. After presenting the entire range of frequencies at 30 db, the sensation level was then decreased to 20 db for a complete sweep, then to 10 db and lesser intensities.

TABLE 1.

PEARSON PRODUCT-MOMENT CORRELATIONS BETWEEN THE AUDIOMETRIC THRESHOLDS OF STANDARD AUDIOMETRY AND THE EAR-CHOICE TECHNIQUE.

	FREQUENCY					
	256	512	1024	2048	4096	8192
Right Ear	.98	.99	.98	.99	.99	.96
Left Ear	.99	.99	.99	.99	.99	.97

e. Each time the subject failed to respond, the threshold for that ear at that frequency was determined by increasing and decreasing the sensation level in five db steps, until a consistent response was obtained. The attenuator was then reset to the original sweeping level and the sweep continued.

f. When a threshold was obtained, that particular threshold was checked by presenting the stimulus once again at the time of the next lower sweep; after this it was eliminated from further testing.

4. If the initial stimulus was inaudible in either or both ears, that threshold was found immediately, and the stimulus level returned to the initial Sensation Level of 30 db.

RESULTS

Of 240 comparisons (20 subjects, 6 frequencies, 2 ears) between audiometric thresholds for the Ear-choice and the Standard techniques, there were fourteen deviations of 10 db and 88 of five db. One hundred thirty three (133) comparisons show no differences between thresholds obtained on the two tests. Thus, 92% of the comparisons fall within the plus or minus five decibel deviation which might reasonably be expected in thresholds obtained by a test-retest using exactly the same procedure both times.

Table 1 presents the Pearson product-moment correlation coefficients calculated between the threshold values of the Standard Technique and the Ear-choice Technique. Each r is of high statistical significance for the number of cases in the sample.

In addition to the product-moment correlation, a double classification, multiple scores per cell analysis of variance was calculated.[5]

TABLE 2.

SUMMARY OF ANALYSIS OF VARIANCE OF AUDIOMETRIC THRESHOLDS FOR 20 SUBJECTS TESTED BY TWO TECHNIQUES (RIGHT EAR).

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARES	F
Between Frequencies	15189.58	5	3037.92	5.72
Between Techniques	3.73	1	3.73	—
Interaction	27.52	5	5.50	
Within Cells	121172.50	228	531.46	
Total	136393.33	239		

A summary of this analysis is presented in Tables 2 and 3. For the right ear the F ratio for between frequencies (rows) using the within cells variance as the error term is significant beyond the 1% level. For both ears the between techniques (columns) variance is actually smaller than the error term. In accordance with this statistical analysis the differences between the thresholds obtained by the two techniques must be regarded as due to chance.

These results seemingly justify the conclusion that the threshold measurements obtained by the Ear-choice technique are at least as good a measure of hearing acuity in individuals with hearing loss as are those obtained with the Standard Audiometric technique.

In the light of these results it seems justifiable to restate some of the advantages of the Ear-choice Technique.

1. The test may be completed in an extremely short time.

2. Motivation of young children seems to be strong in this technique.

3. The Ear-choice Technique has been shown to be at least as accurate a measurement of hearing acuity in normal hearing adults and in adolescents with known hearing losses.

4. The question "Where do you hear it" gives another check on the accuracy of the subject's response.

5. The Ear-choice Technique is very simple to administer.

TABLE 3.

SUMMARY OF ANALYSIS OF VARIANCE OF AUDIOMETRIC THRESHOLDS FOR 20 SUBJECTS TESTED BY TWO TECHNIQUES (LEFT EAR).

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARES	F
Between Frequencies	4231.00	5	846.20	1.59
Between Techniques	17.60	1	17.60	--
Interaction	61.65	5	12.33	
Within Cells	121468.85	228	532.76	
Total	125779.10	239		

SUMMARY AND CONCLUSIONS

A sample of 20 adolescent students with hearing losses was tested by both the Standard Audiometric Technique and the experimental Ear-choice Technique. Ninety two percent of the thresholds differed by five db or less as measured by the two techniques. The product moment r's between the two techniques ranged from +0.96 to +0.99. An analysis of variance was used to compare the thresholds of the two tests and no statistically significant differences were found between the two methods. On the basis of the results obtained by Curry and Kurtzrock and the present study the Ear-choice Technique can be used with confidence both for normal and hard of hearing adolescents and adults. A further study of the reliability and validity of this technique when used with children of lower age levels is now under way.

321 Illini Hall.

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XVIII

EFFECT OF A COMBINED ADMINISTRATION OF AUREOMYCIN AND NICOTINIC ACID IN OZENA

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and

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During the past few years reports published by various authors have indicated the possible therapeutic value of antibiotics in treatment of ozena.

This disease, first described by Fraenkel in 1876 has attracted the attention of many investigators because of the difficulty in its treatment. Various remedies have been suggested: medical, hormonal, surgical and antibacterial, as well as combinations of these procedures, were employed, but none of them with entirely satisfactory results. Specific treatment has not yet been discovered due to the obscure nature of the disease.

While the etiology and pathogenesis of this disease are still controversial, the diagnostic procedures are well defined and offer no serious problem. They are based on clinical, bacteriological and pathological evidences.

Clinically, it is characterized by marked atrophy of the nasal mucous membranes and turbinates, abnormal patency of the nasal fossae, a foul-smelling odor combined with the formation of greenish-yellow crusts; in addition, anosmia is found in most of the patients, who usually are depressed and complain of headache and nasal obstruction in spite of the marked width of the nasal cavities.

The bacteriological flora in this disease is not uniform, although quite characteristic. The etiological significance of the isolated organisms remains questionable, but the constancy of these findings

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is of value in confirming the clinical diagnosis. In 1894 Loewenberg and Abel found in cases of ozena an encapsulated gram-negative bacillus very similar to Friedlaender's bacillus, which is now classified as Klebsiella[1] and which has been repeatedly confirmed by many authors. It is nevertheless doubtful whether it is an etiological agent of the disease. Other micro-organisms, such as a gram-positive bacillus of corynebacterium group, Perez' bacillus (B. foetidus), Ps. aeruginosa and different gram-positive cocci have often been found as well.

The pathological changes consist in replacement of the normal respiratory epithelium by stratified squamous nonciliated epithelium. There is an atrophy of glandular elements and thus a decrease in amount of mucus lubricating mucous membranes. The tunica propria shows diffuse fibrosis and is infiltrated by numerous cellular elements around the vessels and glands. This perivascular infiltration may lead to endarteritis obliterans. Atrophy also attacks the bony structures, particularly that of the inferior turbinates. In more advanced cases all stages of degeneration are evident.[2, 3]

The development of the new antibiotics in recent years suggested their use "in combating this disease or at least a treatment which would allay the unpleasant signs and symptoms of the condition."[4]

Simonton[5] tried the effect of streptomycin in eight patients with ozena with marked improvement in five of them. Thornell[6] added his own seven cases to those of Simonton and noted a marked improvement. On the other hand, Tavani[7] was unsuccessful in the treatment of ozena with stremptomycin, and attributed the bad results to the fact that the nasal flora was resistant to this antibiotic.

Menzio[8] pointed out the beneficial effect of nicotinic acid in cases of both early and advanced ozena. He[9] also showed that intravenous nicotinic acid raises the temperature of the nasal cavity. Cantele and Giuffrida[10] reported good results with nicotinic acid in ozena due to its vasodilatory effect.

In view of the fact that aureomycin has been found to be an effective antibiotic against Klebsiella organisms[11] and considering the low toxicity of this drug, we made an effort to influence the symptoms of this disease, combining aureomycin with a potent vasodilator, such as nicotinic acid; we thus hoped to achieve closer contact of the antibiotic with the affected tissues.

MATERIAL AND METHOD

Our study included 11 patients of whom four were males and seven females, their ages ranging from 11 to 28 years. The range of duration of their illness was from three to eighteen years. The patients were seen daily in the Ear, Nose and Throat Out-Patient Department. Two of them were hospitalized during the whole period of treatment. In all the patients, a number of nasal swabs for cultures were taken at the beginning, and at the end of treatment. Altogether 78 cultures were made. In three cases biopsies were done before and after treatment from the same places of the nasal turbinates. A complete ear, nose and throat examination was carefully performed. Sinuses were x-rayed and in cases of their haziness, proof punctures were performed in order to exclude the possibility of ozena secondary to sinus infection. Kahn tests were performed in all the cases. During the collection of data, attention was paid to the duration of illness, amount of crusts, intensity of fetor, degree of atrophy, the sense of smell, and general subjective signs.

The method of treatment consisted in daily oral administration of 250 milligrams of aureomycin at 6 hourly intervals (1 gram daily). Simultaneously, nicotinic acid was administered orally in doses of 50 milligrams three times daily and in addition 50 milligrams of nicotinic acid were administered intravenously. None of the patients was allowed to use local treatment or nasal irrigations. The duration of treatment was 8 to 14 days.

The estimation of effectiveness of treatment was divided into clinical, bacteriological and pathological changes. On termination of treatment, the patients were re-examined clinically and bacteriologically at first weekly and later at fortnightly and monthly intervals.

RESULTS

The immediate clinical effect of the combined treatment of aureomycin and nicotinic acid in our cases of ozena was very impressive. In a total of 11 cases, it was noted that 10 of the patients had markedly improved both subjectively and objectively. One patient showed no improvement. Out of 10 patients who had fetor before treatment, eight showed complete disappearance of this symptom, one had slight improvement, while two cases showed no change. In all but two of the patients, the crusts disappeared at the end of treatment. Atrophy, which was present in all the patients, and anosmia, (which was complete in five and partial in two) were the symptoms least affected with only three and four patients respectively showing slight improvement. On the other hand, the sense

of nasal blockage, burning sensation and headache disappeared in all the patients but two.

The immediate bacteriological results were the disappearance of Klebsiella organisms in four out of nine cases, and of C. diphtheriae in three out of four cases. The beta haemolytic streptococci disappeared in all three cases where they were present. Detailed bacteriological results are given in the table.

The histo-pathological examinations showed no improvement in two patients while in the third patient no conclusion could be drawn, as she became pregnant.

Contrary to the very satisfactory condition of the patients immediately after treatment, the subsequent results were disappointing. Beginning with the fourth week after the end of treatment, some of the patients returned with similar complaints as previously. Two patients were not re-evaluated, as in the first patient no improvement occurred, while in the other pregnancy intervened. Of the remaining nine patients, six relapsed between one and four months after the end of treatment, while the other three patients were re-examined within two, four and ten weeks respectively, and found to maintain their good condition. Bacteriological follow-ups are given in the table.

COMMENT

Proetz[12] in his "Forty Questions in Otolaryngology" states: "Atrophic Rhinitis: Practically everything about this disease requires explaining; its cause, the nature of the atrophy and, above all, the treatment."

The patients reported in this study have been suffering from ozena for many years. The crusts, odor, the blockage of nose and headaches interfered seriously with their everyday activities. Various treatments have been tried on these patients previously, but without success.

Menzio[8, 9] and Cantelle and Giuffrida[10] reported good results with nicotinic acid in ozena, giving a vasodilatory effect. Lomeo[13] emphasized the advantages of nicotinic acid administered intravenously or orally in ozena.

When this study was near completion, the paper of Sternstein[14] came to our attention. He reported a method for treatment of ozena by combining the local use of antibiotics with vasodilators given orally or parenterally. The antibiotics employed were strepto-

TABLE I.
RESULTS OF CULTURES.

NAME	BEFORE TREATMENT	AFTER TREATMENT	LAST REPORT
S. K.	Klebsiella Coryn. Diphth. Strept. alpha-hem.	Pseudomonas aeruginosa	Pseudomonas aeruginosa
B. O.	Klebsiella Pneumococcus Staph. aureus	Klebsiella B. proteus	Klebsiella Staph. aureus Coryn. pseudodiphth.
D. G.	Strept. beta-hemol. Staph. aureus Coryn. pseudodiphth.	Staph. aureus B. proteus	Strept. beta-hemol. Pneumococcus Neisseria catarrhalis
Z. R.	Klebsiella Staph. aureus Coryn. pseudodiphth.	Klebsiella	Klebsiella Strept. anhemol.
A. A.	Klebsiella Strept. beta-hemol.	Staph. aureus Coryn. pseudodiphth. B. proteus	
Z. H.	Klebsiella Coryn. diphth. B. proteus Strept. beta-hemol. Staphylococcus aureus	Coryn. diphth. Strept. anhem. B. proteus	Coryn. diphth. Staphylococcus B. proteus
M. H.	Klebsiella (poor growth) Coryn. diphth. Staphyl. aureus	Staphyl. aureus	Coryn. diphth. Staphyl. aureus
M. A.	Klebsiella Strept. beta-hemol. Staphyl. aureus Coryn. pseudodiphth.	Klebsiella Coryn. pseudodiphth.	Klebsiella Staph. aureus
H. S.	Staph. aureus Strept. anhemol.	Staphyl. aureus	Staphyl. aureus Coryn. pseudodiphth.
S. L.	Klebsiella Staphyl. aureus Strept. anhemol.	Klebsiella Staphyl. aureus	
N. F.	Klebsiella Coryn. diphth. Strept. anhemol.	Klebsiella	

mycin penicillin, bacitracin and gantrisin. Histamine, priscoline and niacine were used as vasodilators. In his group of thirteen cases, four had striking improvement, six had significant improvement and three showed only slight improvement. He applied the antibiotics locally and nicotinic acid orally. However, no follow-up after treatment is given.

The efficacy of aureomycin "in vitro" against Klebsiella organisms has been demonstrated by Bliss and Chandler.[11] Nataro et al[15] used this drug with great success in a case of acute primary Klebsiella pneumonia in men, and noted that this drug was even more effective than streptomycin in similar conditions. Aureomycin was chosen by us considering its good effect on Klebsiella organisms and above all its low toxicity. This low toxicity compared with the known toxic effects of streptomycin is of high advantage in treatment of this disease, since a prolonged administration might possibly be indicated. The immediate results were most encouraging, but later most of our patients relapsed. However, the fact that there was such an immediate and marked improvement in 10 out of the 11 patients treated seemed very signficant to us.

As the result of treatment, atrophy and anosmia were only slightly changed, but the fetor, headaches, crusts, sense of nasal blockage and burning were very considerably affected. It is this improvement which gives great relief to the patient, frees him from his depression and enables him to mix again in society.

The composition of the nasal flora in our patients corresponded approximately to the findings of most authors. The incidence of Klebsiella organisms in our group of patients was similar to that of Mimica et al,[16, 17] but the percentage of diphtheria organisms was lower. Our results agreed with those of Pierangeli,[18] who found about 30 per cent of diphtheria organisms in his group of 75 patients.

Simonton[5] and Gargani and Faggioli[19] noted diminution of nasal flora after treatment with streptomycin. In our cases, as the result of the combined aureomycin and nicotinic acid administration, the culture became negative for Klebsiella and diphtheria organisms in four out of nine, and three out of four cases respectively. The immediate marked clinical improvement did not correspond entirely with the bacteriological changes. This discrepancy was even more evident in the later results. In view of the small number of cases studied and the unequivocal results, no conclusion could be drawn as to the part these organisms play in causing the signs and symptoms of this disease.

We have recently started a therapeutic trial with a 3 per cent aureomycin ointment. This ointment is used on patients who relapsed after parenteral treatment with aureomycin and on untreated patients. We are impressed by the first results, and this simple treatment seems to us to be promising.

We feel that the period of treatment in our patients might not have been adequate, and it is our impression that a prolonged maintenance treatment may relieve the distressing symptoms of this disease. Further clinical and bacteriological investigations with both parenteral and local aureomycin administration are under way.

It is of interest also to note that three of our patients showed a marked acrocyanosis. The only patient who did not respond to treatment at all, showed evidence of a vascular derangement in her extremities, with scleroderma and Raynaud's disease, as confirmed by capillaroscopy, oscillometry and measurement of skin temperature of the extremities. The local nasal changes in these cases might be a part of a more generalized neurovascular derangement.

Keeping in mind the underlying pathology of ozena which has the character of a prolonged, chronic inflammation, accompanied by vascular obliterative changes, one could hardly expect profound and permanent changes which would reverse the course of the disease and bring its complete cure. The maximum that can be achieved at present is an alleviation of the signs and symptoms of this disease.

SUMMARY

1. A combined parenteral administration of aureomycin and nicotinic acid for the treatment of ozena is presented.

2. Aureomycin was used because of its specificity for Klebsiella organisms and its low toxicity. Nicotinic acid was chosen for its beneficial vasodilatory effort.

3. The estimation of effectiveness of this therapy was based on clinical and bacteriological studies in 11 cases of advanced ozena.

4. The immediate results of this treatment were very impressive and marked subjective and objective improvement was noted in all but one of these patients; however relapses occurred later in most of the cases.

5. It is believed that the period of treatment may have been too brief, and that a prolonged maintenance dose may be indicated to prevent these relapses.

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XIX

NASAL IMPLANTS IN CHILDREN AND IN ADULTS; WITH PRELIMINARY NOTE ON THE USE OF OX CARTILAGE

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During the last five years otolaryngologists have extended and increased their interest in many phases of nasal surgery. A large variety of rhinoplastic procedures, many of which were devised for strictly cosmetic needs have gradually become adopted in the effort to correlate surgery of the external pyramid with intranasal operations in the restoration of nasal function.

Among these is the utilization of implants of inorganic and organic materials and of autogenous and isogenous bone and cartilage.

For the last 60 years the chief indications for the use of these substances were (1) the restoration of contour and (2) the establishment of support. To these we can now add the functional ones of:

1. Repair of nasal roof.
2. Restoration of the hard nasal septum.
3. Narrowing of excessively widened nasal chambers.
4. Repair of deformities of the nasal lobule which includes:
    a. Faulty nasal projection.
    b. Columella deformities.
    c. Collapsed alae.

As yet no single graft or implant material is completely satisfactory for all purposes and so the search for likely substances continues today as it has for the past half century. New plastics such

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From the Department of Otolaryngology, Cook County Hospital.
Read before the Chicago Otolaryngological Society, November, 1951.

as acrylics, polythylene, and polyvinyl alcohol, preserved and refrigerated bone, and ox cartilage have stimulated vigorous research into this subject, a little of which is embodied in this report.

The choice of the best material to be used as a graft or implant depends on a number of variable factors which must be individually assessed. Among these are (1) location and blood supply of the recipient area, (2) type of deformity, (3) the extent of the disturbed function, (4) the nature of the previous nasal surgery or injury, (5) the age of the patient, and (6) the integrity, texture and elasticity of the skin. However, it is not the purpose of this paper to evaluate the various factors which influence the success or failure of a graft or implant but rather to present some experiences with the different types of material utilized.

Bone.—We have previously reported on the use of autogenous cancellous[4] bone obtained from the iliac crest. Where its utilization is definitely indicated it is the most satisfactory graft material of all. We used it in saddle deformities, congenital and traumatic, in patients who had previous surgery with other types of grafts and implants but with resulting failures, and patients with marked loss of nasal bones due to previous surgery, trauma, infection, or developmental deficiencies. Roentgenographs taken two to ten years postoperatively show the presence of the bone graft with firm bony union to the bed and with only slight loss in size of the graft. These results were achieved by (1) preparation of a fresh bone bed on which to place the graft, (2) preparing a cancellous bone graft which was slightly over-sized to allow for about 15% shrinkage by absorption, (3) modification of rhinoplastic technique so that interference with the blood supply was kept at a minimum, and (4) maintenance of immobility of the grafts with stents and adhesive plaster for long periods of time—six to 12 weeks. The use of autogenous cancellous bone is today widely accepted and has very many exponents.

Autogenous cortical bone from the clavicle is easily available and can be used where subsequent complete absorption will not be a handicap.

Preserved Bone.—Preserved homogenous, cancellous bone was used in a series of patients for the following reasons:

1. If successful, it would eliminate the additional surgical procedure of procuring the graft from the ilium.

2. It would reduce the convalescence time and prove less of an economic and physical loss to the patient.

3. It would eliminate entirely the possibility of infections about the pelvic bone.

4. A constant supply would be available.

Preserved bone can be easily trimmed with scissors or knife to fit the desired need. One can readily pass a needle with suture through it and sew it to the recipient tissues to assure immobility.

This bone is very well tolerated but is practically always absorbed within two to four months. It is replaced by a variable amount of fibrous tissue which for contour purposes frequently gives a most adequate result. Hence, it is most useful in minor type repairs of the nasal roof in adults and in the temporizing methods sometimes employed in children. It is most valuable as the temporary scaffolding from which the external nasal pyramid can be suspended if a collapse of the nose into the nasal chambers occurs during a combined rhinoplasty and extensive septum resection or as the result of a severe injury.

Cartilage.—Autogenous and preserved cadaver cartilage have been very widely used in dorsum deformities. When septal reconstruction is being performed the pieces of septal cartilage removed can be reshaped and replaced so that there is little need for employing preserved cartilage in this location. For dorsum contour and for eliminating other nasal defects there is little to choose between autogenous and preserved cadaver cartilage. While it is claimed that autogenous cartilage has a slightly better chance of success this is not great enough to warrant an additional operation to obtain cartilage from the ribs or ears. Absorption of any cartilage is frequently noted.

It might be well at this juncture to interject a word of caution concerning the use of aqueous solution of Zephirin hydrochloride as a preservative for either bone or cartilage. While Vadala[5] has reported this as an excellent preservative our experience and the experience of others have been quite to the contrary. We have had 15 cases of failure in quick succession when cadaver cartilage, bone, and polyethylene preserved in 1-1000 aqueous solution of Zephirin were employed. There occurred localized swelling, redness and fluctuation over the dorsum in each case with rapid liquification of the cartilage which required aspiration. In each case the fluid aspirated was thin, straw colored and contained few cells, and no organisms could be cultured from this fluid. Riggs, Hinderer and Chessen[6] are among those who have had similar difficulty with cartilage and bone preserved in Zephirin. Fox,[6] using bone preserved in 1-1000 aqueous Zephirin solution in two spinal fusion cases reports the appearance of fluctuant masses which were aspirated and from which

again no organisms could be grown. One of us (B.) used 1-1000 aqueous Zephirin to irrigate deep soft tissue lacerations before suturing. In several instances the wounds became necrotic and failed to heal by first intention. In view of all this we feel that the solution of Zephirin has a marked destructive effect on subcutaneous tissues producing aseptic necrosis and should not be used as a preservative.

Ox Cartilage.—There are few reports on the use of ox cartilage as implants in nasal deformities. Stant[2] in 1933 described the use of formalin fixed ox cartilage, which was deformalized in sterile water, for this purpose. Gillies and Kristensen[1] in 1950 described in detail a method of preparation and preservation of ox cartilage in 1-4000 aqueous merthiolate solution. They used this cartilage in 65 cases of nasal defects with only four failures. At the time their article was written they had followed a number of cases for two years and found the results clinically satisfactory. Our own series of cases dates back only six months but even in this short span of time we can see several uses for preserved ox cartilage. Compared to cadaver cartilage, ox cartilage is easier to obtain and the quality can be expected to be more uniform. For our cases, ox cartilage was obtained from Armour Laboratories and had been prepared by the method of Gillies and Kristensen. Briefly the method of preparation is as follows:

"Xiphisternal cartilage is taken from 18 month old heifers at the "kill" floor and placed immediately in 1-4000 aqueous mertthiolate solution. The soft tissues and perichondrium are then dissected free and the cartilage dipped into boiling water for one minute. The cartilage is then transferred to jars containing 1-4000 merthiolate in physiological saline solution. The preserving solution is renewed the following day and once a week thereafter."

At the time of use, the cartilage is removed from the jar and a suitable piece selected. After carving it to the desired shape, it is washed thoroughly in physiological saline solution to remove as much as possible of the preserving fluid. The cartilage is then ready for the final trimming and implantation in the host.

We have used ox cartilage to date in 40 cases for a total of 57 implants. These may be divided into the following groups:

1.	Dorsum implant	27
2.	Columellar implant	8
3.	Lobule tip implant	12
4.	Intraseptal implant	9
	Total	57

In three cases the implant to the dorsum was promptly absorbed. In two dorsal implants there was swelling and slight fluctuation over the graft. After aspiration this subsided and one month later the implants were observed to be still present. In three other patients there was slight swelling over the implants which subsided without treatment and without loss of the cartilage. In two instances the cartilage after three months became absorbed without any previous evidence of incompatibility. In all the other cases the implants appear to be in good condition. Not a single instance of curling or twisting was noted. Thus, to date we have had about 80 per cent successful implants. The poor results occurred in patients who had poor skin, previous surgery in the recipient area, or scarred or otherwise poorly nourished graft bed. We, of course, do not believe that in this short time we can present an adequate test of this material. However, we do know that ox cartilage initially at least, appears to be well tolerated in a large percentage of cases. In view of its availability it merits further consideration and study.

Implants in Children.—The most specific needs for implants in children are (1) the restoration of contour of the roof, especially over the cartilage vault, (2) the stretching of the skin in this area in preparation for future permanent correction, (3) the prevention of subcutaneous cicatrix formation in the dorsum region, and (4) the repair of septum deformities and defects.

For all of these we have used preserved human and ox cartilage with about equal results. Ten to 20 percent absorption of the grafts is to be expected. Autogenous cartilage from the ear is similarly useful. Small implants of plastics and ivory are remarkably well tolerated. To obtain good sized straight non-curling slices of cartilage, we used the crest of the ilium in children. In the very young the iliac crest is cartilage—not cancellous and cortical bone—and slices are easily obtained without endangering the future growth of the pelvis. The fate of this cartilage is no surer or different from other autogenous or preserved cartilage. It is as readily absorbable, but we have never seen it start an appreciable local reaction.

The care of hematomata and abscesses of the septum especially in children requires special mention.

Following the usual drainage instituted in such cases a careful inspection of the septum must be made to determine the extent of the septal cartilage destruction. It will often be learned that much if not all of the quadrilateral cartilage has disappeared and perhaps too the upper lateral cartilages are involved. This leads to saddling

of the cartilaginous vault and retraction of the columella and deformities of the upper lateral cartilages, and the nasal lobule. Replacement of the missing cartilage must be made.

Autogenous cartilage from the ribs, ears, or crest of the ilium can be used. In our experience all of these as well as all other substances are very likely to be absorbed, or extruded. We feel that following an abscess or hematoma the septal tissues are not ready to receive implants for many months, but at the same time if we allow healing to proceed unhindered the fusion of the two mucosal flaps will become so firm with solid scar formation that subsequent separation will be impossible. Therefore, we recommend that at about 8 to 12 weeks following subsidence of the acute infection some material be placed between the flaps even though the benefit may not be permanent. For this we have used fresh autogenous or preserved cartilage. Not one was permanently retained. However, one to two years later we have been able in all of our patients to find a plane of separation and have then reinserted the implants. The value of ox cartilage in these instances has been most noteworthy. One of our ivory implants in an adolescent septum is still intact after 13 years.

Implants in Atrophic Rhinitis.—We reported to this society last year on the use of submucosal bone and cartilage implants to narrow the nasal chambers in atrophic rhinitis. Since then we have used plastic substances for the same purpose and with very satisfactory results. Twelve times we have placed numerous small pieces of polyethylene underneath the lateral nasal mucosa after mobilizing the inferior turbinates according to the method previously described. To date these patients have been markedly improved. We have in ten instances used polyvinyl alcohol—a plastic sponge material—which shows promise of even greater effectiveness. This Material has been used by the Mayo Clinic in chest surgery and in Chicago for orbital implants after enucleation with great success. It is inert, has a capacity to absorb moisture and thus keeps itself soft. Commercially it is available as "Ivanol."

We think this is the first reported use of this substance in the nose and we find that it is being well tolerated and its use in the intraseptal space as well as in the lateral nasal wall may well prove to be a valuable addition to the treatment of atrophic rhinitis.

SUMMARY

1. There are many functional as well as cosmetic indications for using grafts and implants in the nose.

2. No one material is as yet universally adequate.

3. Autogenous cancellous bone when indicated is the best graft material available.

4. Implants of autogenous cartilage from the ribs, ear, and septum, ilium, preserved human bone and cartilage, ivory, plastics and ox cartilage all have a place in this surgery and should be used.

5. Zephirin HCl should not be used as a preservative medium.

6. The use of implants in children is recommended. Their use in the repair of the destroyed septal cartilage following abscess or hematoma to prevent later deformity is imperative.

7. Narrowing of the nasal chambers with extra mucosal implants in the lateral nasal wall and in the septal space is again highly recommended in the treatment of atrophic rhinitis.

Author's Note: Since this article was written we have used ox xipho-sternum cartilage satisfactorily in more than 200 additional cases.

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XX

REFLECTIONS ON THE AETIOLOGY OF THE SIMPLE INFLAMMATORY AFFECTIONS OF THE UPPER AIR-PASSAGES

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[*The pre-antibiotic days though only a decade ago, already seem so remote, and the thinking thereof so obsolete, that it is difficult to realize that even the pre-bacterial days are only the span of a man's life behind us, and that the recognition of allergy as such dates back a mere thirty years.*

A discussion of the etiology of an infectious disease by members of a learned society, all of them unaware of the existence of pathogenic organisms to say nothing of allergens, is recorded in the following presentation reprinted, by permission, from the Transactions of the American Laryngological Association of 1885.

One cannot help being amazed at the elaborate recital of minutiae and the diversity of involved explanations, always missing the main point; nor can one help being forcibly reminded of the similarity between these discussions and some of the symposia of today on subjects of which we, ourselves, still know so little.

How appropriate the Latin quotation with which Mackenzie naively adorns his paper!—Ed.]

* * *

"Ille bene curet, qui bene morborum causas agnoscit."—CELSUS.

In view of all that has been said and written concerning inflammatory conditions of the naso-laryngeal tract, it is amazing to find what little advance has been made toward a more rational conception of the causes that underlie diseases of such prevalence and wide distribution.

It is not the purpose of the present communication to treat in an exhaustive manner the conditions under which inflammation of

the upper air-passages develops, but simply to offer for your consideration a few general observations and desultory reflections, which, I trust, through the discussion which follows them, may serve to throw new light upon the subject, and stimulate others to more perfect and fruitful research.

At the outset of our inquiry we should divest the mind of the idea that the pathology of nasal and laryngeal disease is an isolated pathology. The eruption of catarrhal processes in the respiratory tract is governed by the immutable laws that condition the development and course of inflammation in general, and the rational interpretation of nasal and laryngeal affections presupposes, therefore, the application of general pathological principles to the peculiar conditions which the anatomical and physiological functions of the structures involve. Above all, we should remember that peculiarity of structure is not anatomical isolation; we should remember the correlation of organ and organ, the sympathy of tissue and tissue which make up the perfect physiological life of man. In looking upon the subject from the high vantage ground of general pathology and laws of health, we may, therefore, more readily apprehend the rôle which external and internal influences play in the evolution of nasal and laryngeal disease than if we viewed the same from the level of a narrow specialism or from the standpoint of the mere empiric.

I. Inflammation of the upper respiratory tract, either in its entirety or localized in its individual parts, is a disease of the human race which has existed from the remotest period of historic time.

II. As the chief predisposing and exciting causes of the affection have been in operation for all ages, it follows, therefore, that its origin is coeval with the birth of man.

III. The evolution of nasal, pharyngeal, and laryngeal inflammation in a given locality is, in all probability, a part of its geological history, and goes on *pari passu* with its varying meteorological conditions. Hence the geographical limits of the disease have varied with the different epochs of the earth's formation.

The elaboration of the above propositions involves an inquiry into the origin, the predisposing and exciting causes of nasal, nasopharyngeal, and laryngeal inflammatory affections, and their distribution over the surface of the earth.

ORIGIN.—In the third book of Plato's "Republic" the philosopher tells us that the names of catarrhs were unknown to Homer, and only came into use in the age of Socrates. This assertion of the Grecian sage has been made the groundwork of the thesis that

catarrhal diseases are the products of civilized life, and furnishes, among other things, the basis of the Schneiderian argument that these affections are born of luxury and ease and of the general degeneracy of mankind.

It is doubtless true that a marked tendency to catarrhal diseases belongs to modern man from the accidents which pertain to his environment, and that as civilization awakens morbid conditions unknown or rarely met with in the savage state, so the disposition to inflammatory troubles of the upper respiratory passages may be encouraged by transmitted vices and the enervating surroundings of modern social life. At the same time it is reasonable to assume that, as the chief causes productive of acute and chronic inflammation of the naso-laryngeal tract have been in operation from the remotest times, the origin of the affection is therefore traceable to that of man himself.

The very etymological derivation of the word *coryza* carries us back through the dialects of the Hebrews, Arabians, Chaldeans, and Assyrians to the time when history emerges from fable; the attention paid by the most ancient exponents of medical art, of which we have any record, to inflammatory states of the nose and throat, implies the former frequency of these affections, while the derivation of the terms *angina* and *cynanche,* and the early origin of bronchotomy, point to their recognition of the most dangerous forms of laryngeal disease. The classification and correct clinical history of disease is, moreover, a process of gradual evolution, and, in view of the confusion which reigned among the latter-day nosologists in regard to laryngeal affections, it is not surprising that in the most ancient records of medicine we fail to find that exact anatomical division of catarrhal affections which was the natural outcome of subsequent more advanced anatomical and physiological investigation.

Inflammatory disorders of the upper respiratory apparatus may be the result of a host of conditions external to the body which arise from man's relation to the outer world, may proceed from agencies within the organism, whose sphere of operation embraces the system as a whole, or is limited to its individual parts, or, finally, they may be the outcome of defective anatomical and physiological relations—of absence or abrogation of activity in the respiratory structures themselves and in the functions and forces under their control.

The discussion of the causes proceeding from the first source leads naturally to the consideration of the geographical distribution of the affection.

GEOGRAPHICAL DISTRIBUTION. — *Predisposing and Exciting Causes.*—The geographical limits of nasal and post-nasal catarrhs are as yet imperfectly defined, but it is highly probable that they bear a close relationship to the distribution of catarrhal affections of the respiratory organs in general over the surface of the earth. Thus it may be laid down as a rule that catarrhal inflammation of the nasal passages is much more frequently met with in cold than in warm countries, in high than in low latitudes. The observations of travelers and explorers show that the nearer we approach the equator the less prevalent become affections of the respiratory apparatus, while in the temperate zone they are the most common of all diseases, and preponderate in these regions according to the proximity of the individual localities to the polar circle. In the temperate zones of both hemispheres catarrhs are more frequently met with in those places which lie between the isothermal lines of 18° and 4° (Seitz). The prevalence of these affections is related not only to the geophysical position of a given country, but also to its elevation above the surface of the sea; the higher above sea-level, the more marked the tendency to catarrh (Hirsch, Seitz), a fact partially explicable by the analogy of natural conditions and those of the frigid lations between high mountainous regions and those of the frigid zones. In every zone the geographical distribution of the complaint depends, other things being equal, apparently mainly on climatic influences. *In those countries where extremes of temperature follow each other in rapid succession, where the thermo- and barometrical fluctuations are most sudden and occur with the greatest frequency, and where the material composition of the atmosphere is continually changing, catarrhal affections of the naso-laryngeal tract are most frequently met with. The appearance of the disease seems to depend not so much upon the degree of heat or cold as upon the rapidity and intensity of the change from the one to the other.* In warm countries, coryza and allied affections most frequently prevail during the sudden cooling of the atmosphere by rain-showers, electrical disturbances, or when the heated condition of the atmosphere alternates with dampness and chilliness of the nights. In a similar manner the more or less sudden passage from a dense to a rarefied atmosphere, as in balloon and mountain ascensions, favors the development of nasal congestion and inflammation.

The influence of season in the production of nasal inflammation is simply a part of the greater question of temperature mutations, and will therefore vary with the period of greatest temperature changes in a given year. While spring and autumn furnish perhaps the largest percentage of nasal and laryngeal catarrhs, the coryza

which appears in the summer months, when the air is suddenly cooled or altered by electrical and other disturbances, yields to none in the severity of its symptoms and course.

Of all conditions of the atmosphere the most pernicious to the nasal, and consequently to the pharyngo-laryngeal mucous membrane, is perhaps its saturation with aqueous vapor. Its influence is much more potent in the production of nasal inflammation than that of cold. Indeed, the injurious effect of the latter, *per se,* has been grossly overrated, and, if we examine the subject closely, we will find that there are many other agencies at work whose operation explains the prevalence of these affections in the frigid zones and in the regions of perpetual snow. *The effect of moisture is furthermore intensified by its association with intense cold or oppressive heat.*

It is impossible to overlook the rôle of the *winds,* especially when associated with brusque thermometric changes, in the spread of coryza and allied affections. The strata of air of different temperatures which they bring with them, the moisture, dust (germs?), [Fraenkel had announced the presence of bacteria in the nose in 1873 but only in 1886 demonstrated pathogenic cocci.—Ed.] and, in some instances, the physical shock and pressure which they exert upon the body, as well as the rapidity of evaporation of the cutaneous and respiratory transpiration which they occasion, are powerful agents in the determination of naso-laryngeal catarrh. The terrible effect of the hot wind of the desert upon the throat and nose leads the camel instinctively to bury its nose in the sand until the fury of the tempest is past.

Besides the meteorological relations which condition the geographical distribution of these affections, there are others which pertain to the *geological character of the soil,* to the *configuration of the locality,* and to the *emanations which arise from the surface of the earth.* The two former furnish additional proof in favor of the power of climatic conditions in the evolution of naso-pharyngeal inflammation. It is not within the scope of the present paper to enter into an elaborate discussion of this vast and imperfectly understood question, but it may be said, in general, that the temperature of a given locality will depend, to a certain extent, upon the color of the soil and the presence or absence of vegetation. In some countries—as, for example, in Savoy—the peasants spread dark earth upon the land which they desire to cultivate early, which causes the snow to melt fifteen to twenty days earlier than in other localities (Tortual); it is a well-known fact that the temperature is lowered

and humidity of the soil encouraged by the presence of forests or large tracts of dense undergrowth. The presence of vegetation exerts, too, a remarkable influence upon the chemical composition of the air, and hence upon the development of dissipation of nasal and other forms of catarrh. The noxious exhalations from certain forms of vegetable life probably act as indirect or predisposing agencies in the spread of catarrhal disease, while the presence of others, by purifying and tempering the atmosphere or filling it with certain odors, seems to secure immunity from the affection. The sulphurous air of volcanic regions has been utilized from time immemorial in the treatment of laryngeal affections, and the singular infrequency with which the latter are encountered in places where the air is laden with resinous and balsamic odors has been familiar from the earliest times. The *configuration* of a country enters as a factor in the localization of catarrh in so far as it conduces to exposure to the variations in temperature which have been mentioned above.

There are also a vast number of injurious influences dependent upon *modes of life, dress, imperfect sanitary conditions,* etc., which have been brought forward as the alleged causes of localization of catarrhal affections of the respiratory tract, which, although exercising an undoubted irritating effect, are nevertheless purely secondary and accidental, and have led to crude hypothesis and hasty generalization concerning the essential causes of these diseases.

There are a multitude of conditions which follow as the natural results of imperfect sanitation and professional occupation which act as predisposing and often exciting causes of nasal inflammation. In general, it may be said that residence or work in a comfined or overheated atmosphere, or in one filled with impure gases or floating particles of organic or metallic matter, conduces to the development of the disease. Thus it is well known that artisans who are subjected to a dusty atmosphere— tobacconists, workers in woolen goods, stone-cutters, millers, laborers in chemical works, etc., or in overheated apartments, as, for example, bakers—are thereby rendered more susceptible to catarrhal affections. In addition to the meteorological conditions which prevail in elevated regions, as, for example, the Alps, the finely divided particles of metallic dust suspended in the atmosphere are said to be important factors in determining affections of the respiratory apparatus. The nasal erectile bodies are peculiarly sensitive to the impression produced by certain noxious gases, especially those given off in the combustion of coal, while the furnace heat of the modern dwelling, and the dry, impure air of apartments fed by the majority of coal-burning stoves, and the varying temperatures of the different rooms, create a vulnerability

of the mucous membrane which, in our American cities, constitutes a not unimportant ætiological factor.

In some parts of our country there is a widespread popular belief that *dust* is the chief factor in the localization of inflammatory disease in the naso-pharynx. As there are some who ascribe all diseases to the peripatetic excursions of a vagrant micrococcus, so there are others who see in dust the source of all our ills. [!] While it is undoubtedly true that dust, when accidentally lodged in the naso-pharynx, may give rise to inflammation, I believe that comparatively few cases originate in that way. In some of the Western States the prevalence of large quantities of dust in the atmosphere is supposed to determine the geographical distribution of the complaint; but even here, in estimating the amount of injury done by dust in this case, we should not forget the important meteorological changes that condition its presence in the atmosphere, nor should we lose sight of the fact that these localities are thousands of feet above the water-level, a condition that subjects them more easily to impressions made by sudden variations in the temperature and brings them directly under the dominion of the winds that sweep across the continent from sea to sea. Moreover, when an individual is exposed to an atmosphere filled with dust, the greater portion of the inhaled particles is retained within the nostrils. This is due in a great measure, as I have pointed out elsewhere, to the erection of the turbinated corpora cavernosa, which latter serve, in that respect, a certain teleological purpose. That portion which finds its way into the posterior nares is carried into the lower (not the upper) pharynx, not only by the force of the inspiratory stream but also in obedience to the law of gravitation. When the atmosphere is unusually dense, as in storms of dust, this erection of the corpora cavernosa is often so considerable as to necessitate mouth-breathing, and it is to a large extent in this way that the lower pharynx and larynx become filled with foreign matter. It is also a notorious fact that in the nasal passages themselves the region of olfaction is much less liable to catarrhal inflammation than the respiratory passage. The nasal pharynx is therefore infinitely less liable to inflammation from a dusty atmosphere than either the larynx or lower pharyngeal cavities.

Among the influences which, approaching from the external world, encourage the eruption of naso-laryngeal affections, the chief, and at the same time predisposing, exciting cause, and that which determines the geographical distribution of nasal, naso-pharyngeal, and laryngeal catarrh, is that combination of varying meteorological conditions which are understood when speaking of a changeable climate;

the home of naso-laryngeal catarrh is the land of the greatest and most rapid thermo- and barometrical change.

Turning now from the effect of temperature changes and the direct action of local irritation from substances derived from the external world to the agencies which, operating within the organism, determine the localization of catarrhal disease in the nasal passages and throat, we must confess that the ancients exhibited the greatest shrewdness of observation when they referred these affections to defective digestive processes and lowered powers of assimilation. Catarrhal diseases, according to the fathers of medicine, are due to imperfect "coction"—that is to say, imperfect assimilation—and the resulting discharge or secretion was looked upon as aliment which had not undergone the necessary digestive changes, or, in other words, as half-cooked food. While their notions of the ætiology of catarrhal affections were in the main crude and curiously influenced by the prevailing philosophical vagaries of their time, they nevertheless contain an amount of common sense which it behooves us to pause and consider.

It may be said, in general, that all those influences which impair the general health, interfere with the proper circulation or impair the constituents of the blood, retard the processes of digestion and assimilation of food, or beget a hypersensitive condition of the vaso-motor nervous system, react upon the upper respiratory tract in common with the other organs of the economy, and predispose, other things being equal, to catarrhal inflammation of the same. Thus the latter is more liable to develop in anæmic persons with weak, relaxed conditions of the tissues and who lead sedentary lives, and in those of highly nervous organization, than in those of strong and vigorous constitution and who pass most of their time in active out-door exercise. The existence of syphilis or tuberculosis in an individual is a constant invitation to catarrhal inflammations of the nose and throat, and the same is true in regard to the rheumatic, gouty, scorbutic diatheses, to chronic alcoholismus, and a host of other affections.

Over-indulgence of the appetites, excesses of all kinds, habitual interference with the bodily excretions, notably the intestinal, predispose to inflammatory disease of the naso-laryngeal tract, so that Schneider was not far from the truth when he said that the cure of catarrhs consisted in "sobriety, continuous bodily exertion, and tranquillity of mind."

Besides being predisposed to or conditioned by pathological states of the system as a whole, *catarrhal affections of the nose and*

larynx are not infrequently the result of reflected irritation from its individual parts, as, for example, disease, over-stimulation, or suppression of the functions of the cutaneous (eruptions, suppression of perspiration, etc.), gastro-intestinal (habitual constipation, hæmorrhoids, parasites, etc.), or genito-urinary apparatus (Bright's disease, utero-ovarian irritation, etc.), teeth and gums (caries, dentition, etc.), external auditory meatus or middle ear (inflammation, impacted cerumen, parasites, etc.). Let us examine this subject more closely: As the respiratory passages and skin are the sole avenues through which oxygen reaches the blood, and as they are held in close consent by virtue of their community of function, it naturally follows that the abrogated activity of the one will necessitate vicarious action on the part of the other. This supplementary or compensative action, if prolonged, sooner or later transcends its physiological limits and eventuates in morbid conditions of the organs whose machinery has been thereby overtaxed. Familiar examples of this vicarious action are the congestion or inflammatory disorders of the respiratory apparatus which follow sudden or prolonged interference with the cutaneous functions, as in sudden chilling of the external surfaces or in the exanthemata, the sudden suppression of tinea capitis and eczema, and, on the other hand, the night-sweats of consumption. One word in regard to the eruptive fevers. The catarrhal naso-laryngeal disease may usher in the attack (especially is this the case in measles), may occur coincidentally with the exanthem, or may follow the disappearance of the latter, or, finally, it may not develop until convalescence has begun. The catarrhal affections complicating the essential fevers may disappear completely during the latter period, but in many cases go on to the chronic state. This is especially true, in my experience, in regard to affections of the naso-pharyngeal space. *There seems to be a special tendency to the localization of the disease in this region, and a large majority of the cases of hyperplastic conditions of the adenoid tissue coming under my observation, are directly traceable to some acute blood-poisoning in infancy, as scarlet fever, measles, diphtheria, etc.* This is doubtless due, to a great extent, to the fact that the naso-pharyngeal affection is overlooked by the attendant; but it may be that the *tendency of the eruptive fevers to leave traces of their existence in the glandular structures of the throat* (*notably the tonsils*) *may determine diseased conditions of the adenoid elements of the nasal pharynx.*

It now and then happens that during convalescence from acute infectious processes *irregular fluctuations in the temperature* occur, and even a veritable septicæmic condition, inexplainable by the ordinary examination of the functions of the patient, and which depend

upon the persistence of the inflammatory process in the retro-nasal space. I have observed this after scarlet fever and diphtheria, but there is no reason why it should not occur in other and allied affections. This is an important practical point, for by simply cleansing the naso-pharynx of decomposing secretion (I find bichloride of mercury to be the best agent for this purpose) the temperature becomes normal and the disagreeable symptoms dissipated. In this connection let me observe that in all infectious processes characterized by inflammatory manifestations in the pharynx the greatest relief to the child may be secured through careful cleansing of the retro-nasal cavities. In diphtheria, for example, the greatest comfort is, experienced by attention to this expedient. To digress still further: In the ordinary acute catarrhal throat affections of infancy much comfort may be given, and the tendency to spasm diminished, by careful attention to the nasal and retro-nasal cavities. There is an old woman's saying in this part of the country that "if a croupy child sneezes, it is well." But no one ever thinks of the nasal passages in connection with a croupy infant. Yet there is, nevertheless, always more or less inflammation of the post-nasal passages, and the tendency to spasm may be diminished by cleansing the passages of mucus especially before the child retires for the night.

There is an affection of the skin which is observed in connection with certain forms of coryza, and notably that of sympathetic origin, or rhinitis sympathetica. I refer to urticaria. It appears and subsides with the coryza, and seemingly depends upon the imperfectly defined neurosis or vaso-motor influence which is probably the connecting link between the two affections (possibly some functional derangement of the cervical sympathetic).

Passing now to the *reciprocal relationship existing between the turbinated nasal tissue and the auditory meatus and middle ear,* it is not an uncommon matter in my experience to find that the subjects of chronic nasal inflammation suffer from a more or less constant dryness and itching of the former or a tendency to the inspissation of cerumen, and this, apart from existing disease of the middle ear. I have on several occasions adverted to the rôle of congestive conditions of the erectile tissue in the production of middle-ear affections (doubtless through reflex influence). It remains for me to call your attention to the fact that unilateral coryza, either acute or chronic, sometimes depends upon irritation of the external auditory passage. Two years ago I was consulted concerning a case in which a severe unilateral discharge, with stoppage of the nostril, hemicrania, and other phenomena referable to the same side, of a number of weeks' duration, was completely dissipated by the removal of a mass

of impacted cerùmen from the ear of the affected side; and recently a similar case has come under my professional care in which the swelling of the erectile tissue disappeared upon removal of the ceruminous plug. In the presence of these facts the conclusion is irresistible that an intimate physiological relationship exists between the nasal cavities and the auditory meatus.

It has thus been shown *that nasal and laryngeal inflammation may proceed from the direct or indirect (reflex) irritation of a host of substances derived from the external world, from an almost indefinite number of pathological conditions of the system as a whole, or from irritation or disease of organs distant from the seat of local inflammation.* The predisposing influence of *certain structural peculiarities of the nasal chambers* remains to be briefly adverted to. These consist most commonly in deflection (or malposition) and perforation of the septum, anomalous conditions of the turbinated bones (hypertrophy, abnormal position, atrophy?), and disturbance of the anatomical relations of the nasal fossae either through accident or disease. There are certain anomalies of structure of the throat and nasal passages that are seen in several members of the same family which are undoubtedly inherited and which are of such a nature as to give rise to no inconvenience, or, on the other hand, encourage the development of inflammatory processes; in the latter cases their influence is purely mechanical. There also exists in some families a *peculiar vulnerability of the mucous membrane* of the nose and throat which is sometimes conspicuous for several generations. Such persons are said to "inherit" catarrhal inflammation or to be the subjects of the "catarrhal diathesis"—a view which has descended to us chiefly from the earlier French physicians. It is undoubtedly true that the children of parents debilitated by disease, excesses, or other causes, or who inherit, for example, the enfeebled constitution of the syphilitic or tubercular, diatheses well known as predisponents to catarrh, may, by virtue of the inheritance of a vice of constitution, yield more easily than those of healthy parentage when exposed to the exciting causes of the disease; but there is no evidence yet adduced that puts beyond a reasonable doubt the descent of a simple inflammation from father to son. These remarks apply with equal force to the so-called catarrhal diathesis, which latter may be looked upon simply as a generic term for a multitude of varied physical peculiarities, each susceptible, upon close analysis, of reference to a definite and tangible cause, or to a combination of injurious influences.

Catarrhal inflammations of the nose and throat in the newly born, when not due to gonorrhœal inoculation, probably owe their

origin to causes operating during intra-uterine life. It occasionally happens that inflammatory affections of these cavities are *ushered in at some physiological epoch, as puberty, or existing disease dissipated by the nutritive changes which occur at that period.* The subacute laryngitis which occurs at puberty occasionally develops into a chronic inflammation, especially in the subjects of inherited constitutional vices—a fact which is well to bear in mind both in a prophylactic and prognostic point of view. Inflammatory conditions of the throat and nasal passages occasionally make their appearance at the menstrual period, appearing either coincidentally with the uterine hæmorrhage or as the vicarious representative of that process, and I have seen one case where a catarrhal affection of these passages ushered in the menopause and subsided with the termination of menstrual life.

AEtiology of Pharyngeal and Laryngeal Inflammation.—The chief predisposing causes of acute pharyngo-laryngeal inflammation are the existence of chronic hyperæmia or inflammation of the nasobronchial tract, abnormal state of vitality from inherited or acquired disease, excesses, subjection to imperfect sanitary conditions, and constant confinement to a vitiated atmosphere. While in the vast majority of instances acute inflammation of the pharynx or larynx occurs as a complication of acute or chronic naso-pharyngeal (or bronchial) catarrh, it may nevertheless be met with as a primary affection. I do not share the extreme view of my friend Dr. Bosworth, who has written so well upon this subject, that acute laryngitis only occurs as a symptom of the chronic form. While I regard the existence of the latter as its most prominent predisposing cause, it is nevertheless true that the disease may appear as a primary affection limited to the laryngeal structures.

Apart, then, from the inflammation resulting from local pathological processes, mechanical or chemical injury, abuse of the forces of expiration and inspiration, the isolation of this disease in the larynx (acute primary laryngitis) is one of the rarest of pathological events. In adult and infant it most commonly occurs as a complication of acute nasal catarrh or as a part of a general inflammatory condition of the naso-bronchial tract. I am inclined to believe that in the irritable state of the nasal tissues, and notably the cavernous bodies, resides an important ætiological factor in the adductor spasm which characterizes the disease in the infant; the engorgement of the sensitive area when the recumbent posture is assumed, and the gravitation of the nasal secretions into the laryngeal vestibule, being the most important agents in awakening the reflex laryngeal spasm. In the adult, acute catarrh of the larynx is a relatively rare disease, a

fact which is remarkable, as Flint has pointed out, in view of the frequency of acute pharyngeal inflammation, and illustrates the conservation of the natural law in regard to the extension of inflammation.

The reflex or collateral hyperæemia of the larynx which is present in inflammatory conditions of the nasal and pharyngeal cavities is too often mistaken for acute inflammation, and confusion too often arises, especially when the laryngoscope is not available, from failure to remember the simple truth that hoarseness is not laryngitis.

Chronic catarrhal laryngitis as an isolated affection is rarely met with; it is almost invariably secondary and associated with inflammatory disease of the nose or nasal pharynx, upon which it, in the large majority of cases, depends. Indeed, setting aside the inflammation which results from purely local irritation, it may be laid down as a law that the vast majority of cases of catarrhal, pharyngeal and laryngeal disease originate primarily in inflammation of the nasal cavities. Catarrhal rhinitis leads to inflammation of the pharynx and larynx in one or all of the following ways:

1. By mouth breathing, which I may say acts not only through the irritation of the cold, dry, and impure air inspired through the mouth, as in nasal obstruction, or through the nasal passages, as in atrophy of the turbinated structures, but also by crippling the respiratory and vocal forces, shortening both inspiration and expiration, compelling rapid respiration and resulting vocal and respiratory fatigue.

2. By the constant endeavor to overcome the loss of nasal power and resonance, and the consequent pharyngeal and laryngeal fatigue.

3. In certain cases, by interference with the normal motility of the palatal structures.

4. Through reflected irritation.

5. By the irritation of the atmosphere, vitiated in some instances, not by virtue of its passage through the mouth, *but through the nasal chambers themselves.*

6. By so-called extension of inflammation.

7. Possibly by irritation of secretion.

In studying the pathological conditions of the naso-pharyngeal space and middle ear, we may, for practical purposes, regard these cavities as accessory to the nasal chambers, so intimately interwoven

is their pathology with a diseased condition of the nasal fossæ. As stated elsewhere, in a large proportion of cases of so-called middle-ear inflammation the latter "is merely a symptom of nasal catarrh, and gradually disappears without special treatment upon the removal of its primary cause." When the middle-ear affection is thus symptomatic, it is generally traceable to mechanical causes or to reflected irritation.

Inflammatory conditions of the naso-pharyngeal cavities are encouraged and existing disease of these structures perpetuated by paralytic conditions or defective muscular power (*e. g.*, from existing chronic inflammation, enlarged tonsils, defective innervation, etc.), or from abnormal approximations of their walls (from adhesions). As a consequence of the impaired functional exercise of the structures thereby induced, congestive and catarrhal processes develop in the laryngeal cavity, which result from the constant endeavor by abuse of the expiratory forces to overcome the loss of power in the pharynx. Both loss of power and adhesions, which latter act by crippling the muscular action and disturbing normal anatomical relations, tend also to prevent voluntary cleansing of the retro-nasal space, and thus form another factor in the persistence of the chronic inflammatory process.

Finally, I wish to observe that in a large proportion of cases it will be found, upon careful examination, that the existence of the nasal, pharyngeal, or laryngeal affection is not due to any one particular cause, but to a combination of injurious influences—the resultant of a number of internal and external forces.

DISCUSSION

DR. J. SOLIS-COHEN: We gather from the essay the fact, not fully appreciated, that many catarrhal affections are due to climatic and constitutional conditions, and that this indicates a feature in the treatment which is too frequently ignored. Climatic conditions can not be altered, but the susceptible individual can be protected by suitable hygienic and dietetic supervision. The function of digestion and the secretions of the intestines and kidneys must be attended to, the skin be kept in good order, and thus revulsive measures be adopted to withdraw morbid influences from the upper respiratory tract. Too great an attention to local measures without due consideration to constitutional conditions does not fulfill the requirements.

Acute laryngitis, in my experience, occurs frequently in individuals who are not at all suffering with chronic catarrhal disease, and subsides without such a sequel. On the other hand, subacute inflammations occur most frequently, in my experience, in the subjects of chronic disease as exacerbations due to some special exposure.

The spasmodic affections of infants alluded to in the paper as occurring in the subjects of chronic naso-pharyngeal catarrh are similar in origin to the night-cough of infants, being due to trickling of mucus into the larynx. The indi-

cation, then, is to place the sleeping infant in a position which will not favor the entrance of secretions into the larynx, and, of course, to treat the catarrhal disease.

Dr. H. A. Johnson: Naso-pharyngeal catarrh is common around our lakes, and is frequently benefited by a change of climate to the Western plains, up to an altitude of five thousand feet. In the high mountains the disease is aggravated. In the treatment of this trouble tonics are usually of great value, and we are tempted to overlook the general conditions in the presence of the local lesion.

Dr. Glasgow: I can not strictly agree with the speaker in the relative importance of atmospheric changes and dust in producing catarrhal diseases. Although rapid changes of atmosphere lead to catarrhal disease, I fully believe that dust or atmospheric dirt holds the most important place in their production. We find evidence of this in the fact that people living in atmospheres laden with dust are particularly liable to catarrh. For instance, persons living in my own city, St. Louis, are especially prone to these troubles. Removal into the purer air of the country invariably lessens the difficulty, and it is again aggravated by a return to the city. I believe this is the general experience in all dust-laden cities.

We also see evidence of the importance of dust in the experience of people in the alkali regions of the West, and this without special regard to the elevation. Catarrhal disease is the rule in this section. If previously existing, it is aggravated; if the naso-pharyngeal membrane was previously healthy, a commencing catarrhal inflammation is soon noticed.

Dr. Ingals: I agree with the other speakers as to the necessity for attending to the constitutional condition of the patient, but we must not lose sight of the constitutional manifestations due to the nasal disease. In many cases great impairment of the general health, which may have existed for years, is dependent upon nasal obstruction, and remarkable improvement will immediately succeed removal of the nasal affection.

Dr. Allen: The study of catarrh should be considered in great part as a phase of morphology. The careful study of each separate case is important. It is always well to treat individuals rather than diseases. The identification of the local structural cause of the catarrh is, in the majority of instances, alone possible after unremitting scrutiny.

Dr. Mackenzie: I fully agree with Dr. Allen as to the importance of local treatment; at the same time it should not be forgotten that in many instances the balance between cure and failure may be easily turned by neglect of constitutional measures. Cases occur in which local measures must be carried out before relief can be obtained, while, on the other hand, constitutional measures must be employed in order to secure the maximum of good in treatment. Dr. Glasgow misunderstands the position which I took in regard to dust as a factor in the production of catarrh. My proposition is simply this: That the localization of catarrhal processes in the retro-nasal space or the geographical limits of the disease have little to do with the presence of dust in the atmosphere. While it is true that dust accidentally lodged in the naso-pharynx may give rise to inflammation, I believe that few cases originate in this way, for reasons which I have explained in my paper.

Clinical Notes

XXI

LARYNGOSCLEROMA

REPORT OF A CASE

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Rhinoscleroma is a disease affecting the upper respiratory passages, usually involving the mucosa of the nose and larynx, with granulomatous tissue to the point of obstruction. It is usually found in persons of central or southern European origin, although more and more cases are being diognosed in native-born Americans, especially in the Southwest.

Although comparatively rare, it is nevertheless a slowly debilitating condition, and occasionally, if the laryngeal airway is completely occluded, fatal.

It can be identified by direct culture of a Klebsiella organism known as the Frisch Bacillus, or on biopsy by large foam-like cells called Miculicz bodies.

Originally, the treatment of choice was roentgen therapy, which did not always eradicate or in some cases, did not improve the condition. More recently, streptomycin and dihydrostreptomycin have been used with strikingly good results.

Canfield[1] first reported a case of rhinoscleroma in a native-born person, cured by x-ray therapy. He felt it hastened the end point of sclerosis. Cunning and Guerry[2] reported 102 cases in the United States and Canada, sixteen native born. New et al[3] reported in 1947 the first apparent cure using 95 gm of streptomycin over a two to three month period. Miller,[4] in 1949, reported three cases in Los Angeles successfully treated with streptomycin. Olson[5] reported three cases in 1950, treated by surgical excision plus x-ray therapy, the dosage being 550 roentgen units, in five weekly treatments. Curkovic[6] added another three cases eradicated by streptomycin.

Most recently, Morwitz[7] reported rhinoscleroma cured by dihydrostreptomycin. Eisenstodt[8] reported a case of scleroma of the nose and lips apparently dormant after electrocoagulation and potassium iodide therapy.

REPORT OF A CASE

B. B., a 40-year-old, American-born zinc miner, of Hungarian descent was first seen on May 14, 1951. He was referred by his family physician, because of intermittent hoarseness and dyspnea, of at least four years' duration. In the past week, he had lost consciousness on exertion, at which time he became completely apneic. He denied weight loss, dyhphagia, cough or pain.

His past history is notable in that he had a nasal operation ten years previously by an otolaryngologist in his home town. This gave him no relief from his complaint of nasal obstruction.

His family history was highlighted by the fact that his mother, a native Hungarian, had similar nasal obstruction, but had no treatment, and died of unrelated causes.

Examination of his ears, nose and throat revealed the following findings: The anterior septum had a 1.5 cm perforation, and both vaults were almost completely occluded with scar tissue. Both middle turbinates appeared to be absent. Indirect laryngoscopy revealed a 2 mm chink at the posterior commissure. The remainder of the glottic space was completely occluded by a firm, red, granular mass, involving both false and true cords. His dyspnea was marked. His phonation was almost nil.

He was immediate hospitalized, and a tracheotomy performed, followed by a direct laryngoscopy and biopsy. The report on the latter was as follows: "The tissue submitted for examination, shows groups of large foam-like cells, showing a very distinct cell membrane, typical of Miculicz cells. It is diagnostic of scleroma."

He was therefore started on 1 gm of dihydrostreptomycin daily, and this was continued for 94 days. After one month of therapy, he was able to tolerate his cannula plugged, when there was no exertion. After two months, he kept it plugged twenty-four hours a day, but did no heavy work except cutting the lawn. He was decannulated after 94 days of treatment and returned to his former work without difficulty. At his most recent examination, (March 1952) he reported he sleeps quietly at night, that he has no limitation of physical activity, and volunteers that he can breath through his nose once more. The latter fact is not too well substantiated by the

internal appearance, however. His hearing has remained unchanged and within normal limits, in spite of the prolonged course of therapy with dihydrostreptomycin.

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XXII

CIRSOID ANEURYSM OF THE LIP

MARTIN HERBERT ZWERLING, M.D.

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AND

CDR. RAYMOND GRAVES, (D.C.) U.S.N.

A cirsoid aneurysm is defined as a dilation and tortuous lengthening of a blood vessel due to an arteriovenous fistula. The mechanism of its production has been outlined by Cecil.[1] He explains that the arterial blood, following the path of least resistance, flows directly into the vein instead of through the corresponding capillary bed. The thin-walled veins become distended by the pressure transmitted from the artery through the fistulous opening. These veins eventually become prominent and cirsoid. There are two types of cirsoid aneurysm: The congenital and the traumatic. Dandy[2] states that the percentage of congenital aneurysms far exceeds that of the traumatic type. A review of the literature for the last twenty years discloses no report of a cirsoid aneurysm of the lip published in the United States. The following case is reported because of its rarity.

The patient, aged 20 years, was admitted to the U. S. Naval Hospital, Charleston, S. C., because of a large disfiguring, pulsating mass on his lower lip. He dated the onset of this affection to the age of nine years, when he fell and his top teeth went through his lower lip. Following the injury, slight swelling of his lower lip appeared. The patient engaged in boxing and football between his fourteenth and eighteenth years, and traumatized his lower lip many times. At the age of fifteen years the mass began to pulsate for the first time. Since then, it has become progressively larger and the pulsations more marked.

Examination showed a soft red mass of the lower lip measuring three by three centimeters. The mass pulsated strongly and synchron-

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ously with the radial pulse. Compression of the inferior labial arteries bilaterally stopped the pulsation. A thrill could be felt over the mass but no bruit could be heard. The diagnosis was cirsoid aneurysm of the lower lip (traumatic).

Operation was performed on July 24, 1952. The mass was excised through a V-shaped incision measuring 3.5 centimeters. Bleeding was profuse but was controlled by compression of the lateral margins of the lower lip. The edges of the wound were made slightly concave and following its closure, no pulsations were noted. Postoperatively, the wound healed rapidly and no mass or pulsations recurred. However, the patient complained of a tight lower lip which interfered with eating and talking, and an indentation of the skin of the chin was present.

A second operation was performed on August 27, 1952. An Esser inlay graft was placed in the gingival-labial sulcus. The technique consisted of removing a split skin graft measuring fifteen thousandths of an inch in thickness and 1.6 by 2 inches from the right upper arm. The graft was sewed around a piece of stent with the raw side outward. A pocket measuring 1.2 by 0.8 inches was made in the lower gingival-labial sulcus and the skin graft and stent were buried in the pocket. The incision in the sulcus was then closed.

A third operation was performed on September 5, 1952, at which time the incision in the sulcus was opened and the stent removed. The skin graft was well adherent to the surrounding tissues and the sulcus depth was increased by about 0.8 inches. Postoperatively the indentation of the skin was almost completely relieved and the patient was able to eat and talk normally. The tightness of the lower lip was completely relieved.

Cdr. F. J. McMahon reviewed the sections of the specimen and reported finding a large elongated venous channel with moderate to marked thickening of the wall and ramifications extending radially into the surrounding tissue. Around this were many small, thick-walled venules and capillaries embedded in fibrous tissue. The diagnois was confirmed. The patient was well three months after the extirpation.

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XXIII

BILATERAL ABDUCTOR PARALYSIS

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The problem of double abductor paralysis has received a great deal of attention since King in 1939 made his notable contribution to this subject.

King,[1] in his first report, thought that the transposition of the omo-hyoid muscle to the arytenoid cartilage aided in pulling it outward. Subsequent observations by him and other surgeons proved that the use of this muscle was neither necessary nor advisable.

Kelly,[2] in 1941, described the results of his experience with a window in the thyroid cartilage through which the arytenoid was grasped and fastened to the thyroid cartilage.

McCall and Gardner[3] added to this procedure by the use of transillumination of the larynx to aid in locating the arytenoid.

Orton advised removal of part of the thyroid cartilage at the posterior half in order to expose the arytenoid.

Woodman[4] recommended partial arytenoidectomy with retention of the vocal process and fixation of the cord to the inferior cornu of the thyroid cartilage.

Thornell[5] presented the intra-laryngeal approach for arytenoidectomy.

Other modifications of this operation have been published.

This report deals with seven cases in which the intralaryngeal approach was used. There were six cases resulting from thyroidectomy and one from poliomyelitis. The ages ranged from 23 to 68. In the six females the condition followed thyroidectomy—in the male (aged 23) poliomyelitis. The poliomyelitis patient had been wearing a tube for two years. The post-thyroid patients had been wearing cannulas from 2½ to 8 years.

Read at a meeting of the Los Angeles Society of Ophthalmology and Otolaryngology, September, 1952.

Strange as it may seem, three of the post-thyroid patients had not worn tubes. These patients suffered from lack of them previous to operation for three, four and five years, respectively.

The technique of this operation is carried out through the suspension apparatus. If the patient is not provided with a tracheal tube, one should be inserted previous to operation on the larynx.

We use Pontocaine 2% applied locally to the pharynx and larynx. Pentothal sodium and curare are employed for general anesthesia after which the suspension laryngoscope is placed in position. An injection of two or three cubic centimeters of 1% novocaine with adrenalin is made into the arytenoid area. This will minimize bleeding. An incision 1 cm in length is then made over the arytenoid cartilage extending anterolaterally into the aryepiglottic fold. A laryngeal grasping forceps is used to secure the upper part of the aytenoid. A dissector is then inserted to free the arytenoid of its attachments. The dissector is used laterally first and then by turning the cartilage in the forceps, the attachments on the medial side are released. The cartilage can then be removed by means of rocking movements to separate it from the articulation with the cricoid. Following removal of the cartilage an electrode is inserted into the space and the lateral aspect of the wound is cauterized. The cauterization should be made deeply in the incision lateral to the vocal cord. The scarring which results from the cautery helps to pull the cord away from the midline, although in cases in which the cautery has not been used the results have been good.

An acrylic obturator in the shape of an intubation tube can be placed between the cords at the end of the operation to hold the cords away from the midline. This obturator can be fastened in place with a cord attached to the cheek above and to the tracheal cannula below. We have used the obturator in only three cases.

The operation proved successful in all but one case. This patient was operated upon two years ago but the airway was not adequate. She still wears a tube.

In the others the tracheal cannula was removed in three or four weeks, after it was established that an adequate airway was present. It was made clear to each patient before operation that there would be some impairment of the voice. None of them complained following surgery.

We realize that the complete restoration of function of the larynx is impossible in this condition. We aim to restore the lumen, and in order to do this, some impairment of the vocal function must

follow. I think it is agreed that an opening one-half centimeter in width is adequate for most people and a larger lumen may produce too much change in voice.

In conclusion, this operation offers a simple method of obtaining a lumen in these distressing cases. I believe that it can be recommended as one of the modifications which gives good results in most cases.

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XXIV

ABSCESS OF EPIGLOTTIS

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The following case report of acute abscess of the epiglottis is presented because of its fulminating onset with severe respiratory obstruction and swelling of soft tissues of the neck. Tracheotomy was of paramount importance in therapy.

In 1878 Michel[5] labeled infection of epiglottis, Angina Epiglottidea Anterior, stating that the acute inflammatory process is confined to the lingual surface of the epiglottis though the other parts of the larynx may sometimes be affected to a slight extent. In 1900 Theisen[1] suggested the term, Acute Infectious Epiglottitus. Few papers on this subject are in the literature. During the past half century, not more than 60 cases have been reported in this country. In 1940 Brown[8] classified acute inflammation of the epiglottis in three groups: 1, acute diffuse inflammation, 2, acute ulcerative inflammation, 3, acute inflammation with abscess formation. He reported seven cases which illustrated the above groupings.

Streptococcus hemolyticus and Streptococcus viridans, Pneumococcus, staphylococcus, albus and aureus, are the most frequent invaders. Miller[11] stated that hemophilus influenzae type B, affects the epiglottis in children. The onset is extremely rapid. The course of the infection is fulminating, usually less than ten hours. This organism can also affect the adult, with the same acute symptoms. Brewer[10] reported six cases due to hemophilus influenzae, type B., four of the six cases being in adults. The following case report shows the same clinical picture in an adult as that in Miller's children.

The etiology is still controversial. Theisen,[1] Lederman,[2] Key,[14] Tawse,[16] Miller[11] believe it is a primary infection; Scal,[15] Jackson,[13] Morrison,[17] and Brown[8] consider it to be primary or secondary to adjacent infection or trauma. Most authors point out the acute onset

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of the disease with fulminating respiratory distress, associated with difficulty in swallowing, but very seldom with hoarseness. Diagnosis can easily be made by direct examination, using a tongue depressor. In some cases there may be yellow spots under the surface of the epiglottis. The striking symptom is acute respiratory obstruction. As for treatment, in mild cases, antibiotics under close observation may be all that is needed. In severe cases, tracheotomy should be done immediately. Repeated scarifications are performed in the early stage by some surgeons. Incision and drainage may be indicated when the abscess is obvious. Antibiotics are essential. Brewer[10] (1948) recommended the use of streptomycin especially for the hemophilus influenzae, type B. infection, and thinks it is specific with definite value for this kind of infection. Antiserum may be also worthwhile.

REPORT OF A CASE

Patient, A. C., a 32 year old white stockman, had been first examined by a medical intern and a diagnosis of angioneurotic edema of the larynx had been made. One-half cc of adrenalin (1:1000) was given, but the condition did not improve. The patient had been well earlier in the day. He had been working in the morning and had had a good luncheon about noon. At 2 P. M. he felt a little soreness on the right side of the throat, which increased rapidly and there was difficulty in breathing and swallowing at 5 P. M. At 9 P. M. he had gone to another hospital because of his increasing respiratory difficulty. Unfortunately, he did not see a doctor in that hospital and was told by a nurse to return to the clinic the next morning.

On examination by us at midnight the patient appeared acutely ill and anxious. He spoke with considerable difficulty but there was no definite hoarseness. His breathing and swallowing were greatly embarrassed, so that he had to be in a sitting position. His chest retracted with respiration and he had episodes of spasmodic gagging. Repeated attempts were made to clear the throat and expectorate. His face was pale and cyanotic and he perspired profusely. The rectal temperature was 101 F, pulse 100, and respirations 26 per min. There was marked swelling of the neck, more prominent on the left side. The left submaxillary and superficial cervical lymph glands were enlarged and tender. The floor of the mouth was soft but there was swelling in the hyoid region and he had difficulty in opening his mouth. The throat was edematous around the left tonsil as well as on the retropharyngeal wall which was pale. The mucous membranes of the right side appeared normal. Indirect laryngoscopy

showed a swollen mass located in the hypopharynx and anteriorly in contact with the root of the tongue. The surface was intensely red and shiny. It was impossible to obtain a clear view of the glottic region due to the edema of the epiglottis.

Tracheotomy was immediately performed with the patient in a semisitting position. The respiratory obstruction was relieved and the patient was able to lie flat in bed, but could not take any fluids.

Post-operative medication consisted of 400,000 units of penicillin daily, intravenous fluids, hot compresses locally, and routine post-tracheotomy care.

Laboratory findings: WBC 14,600 per cmm, Hgb 13 grams or 89%, hematocrit 42, differential count—seg. 95, lymph. 5, Wassermann and Kahn tests were negative. Hemophilus influenzae was isolated from the epiglottic region.

X-ray of the cervical region on admission showed the bony structures essentially normal. There was a distinct abnormality of the prevertebral soft tissues and a sharply defined mass (abscess) in the region of the epiglottis, obliterating the valleculae. The mass appeared to descend into the supraglottic region. There was some thickening of the retrotracheal soft tissues, suggesting an extension of the inflammatory process to this area.

In the antero-posterior view, the trachea and larynx were seen displaced to the right and the trachea was markedly compressed.

Hospital course: On July 24, 1952, the patient complained of some pain in the anterior chest, but no emphysema or any inflammatory signs could be found. Indirect laryngoscopy showed thick yellow pus draining from the right upper part of the swollen epiglottis. The abscess had ruptured spontaneously. Penicillin was continued and dihydrostreptomycin 0.5 gm was also started daily. Intravenous fluids were continued for two more days, as the patient still continued to have difficulty in swallowing.

On July 25 he started to drink fluids. There was no further chest pain. The swelling of the neck slightly diminished and the patient slept well.

X-ray on July 25 showed the tracheotomy tube in situ. There was still the marked distortion and swelling of the laryngeal and supraglottic areas, suggesting an inflammatory process here. The reduction in the size of the pretracheal soft tissue suggested early resolution of the inflammatory process in this area.

On July 26 the condition was much improved and he was able to take a soft diet.

On July 27 temperature was normal. The patient ate well and swelling of the neck was markedly reduced. Indirect laryngoscopic examination revealed the epiglottis still moderately swollen and congested. The arytenoids and both vocal cords were normal. On July 29 the tracheotomy wound was corked, and on the following day, decannulation was done.

On July 31 the epiglottis showed slight swelling and the glottic region could be easily seen. Penicillin and dihydrostreptomycin were discontinued. The total dosage of penicillin was 3,600,000 units and dihydrostreptomycin 4 grams. The patient was subsequently followed in the out-patient department. Complete recovery was uneventful.

COMMENT

The literature describes the development in this type of case in adults in less than 20 hours from the onset, in children less than 10 hours. The onset in this case preceded the distressing symptoms by only three hours. The soft-tissue-swelling about the neck and the pretracheal region was due to secondary tissue reaction from the infection of the epiglottis, and subsided as the abscess of the epiglottis drained and healing ensued.

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XXV

CYST OF THE PYRIFORM SINUS

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Numerous reports of cysts of the larynx have been published in recent years. Classification depends upon their location and their origin. Most commonly they are found arising on the inner aspect of the larynx or its aditus. Epiglottis cysts are seen most frequently; then cysts on the cords, the lateral wall and finally on the aryepiglottic folds. Various theories have been brought forward to explain the formation of cysts in this location and in a previous publication[1] they are expounded at length.

Cysts are acquired or congenital. Acquired cysts are due either to direct trauma with transplantation of epithelium into deeper layers,[2] or to inflammatory occlusion of excretory ducts of glands. Manifold are the explanations for the appearance of congenital cysts: displacement of embryonic cells, branchiogenic origin or the meeting of several germinal layers. No valid argument has been advanced to explain the sudden growth from an anlage.

REPORT OF A CASE

A white woman, 52 years old, was referred for difficulty in swallowing, lasting about 4 weeks. Ear, nose and throat examination was essentially normal except that on mirror examination of the larynx a mass was noted in the right pyriform sinus. It filled the posterior portion, was grayish in color and grapelike, with a smooth surface. It rose with movement of the larynx and on phonation. The inside of the larynx appeared normal and muscle function and phonation were not impeded. There was no obstruction of the lumen of the larynx. The diagnosis of cystic tumor of the pyriform sinus was made and the patient consented to surgical removal. This was done under general anesthesia December 13, 1951. A laryngoscope was introduced and the mass brought into view. It filled the lumen of the 'scope. Manipulation revealed that a short pedicle attached it to the posterior surface of the right arytenoid. It was

Presented at the Fall meeting of the Eye, Ear, Nose, and Throat Section of the Connecticut State Medical Society, October 29, 1952.

ovoid, smooth, and glistening. Attempts to remove it *in toto* failed and a small amount of viscous material was aspirated, the sac and the pedicle were then removed. The patient noted slight soreness for a few days, but has been free of symptoms since. Histologic examination of the cyst wall showed it lined by a layer of stratified squamous epithelium overlying a delicate fibrous tissue stroma (McAdams). The diagnosis was epithelial cyst of the pyriform sinus.

COMMENT

Eggston and Wolff[3] describe simple dermoid cysts lined with stratified squamous epithelium without cutaneous appendages which are occasionally found in the pharynx and the larynx. Usually dermoid cysts are filled with caseous material, and their subepithelial tissue contains sebaceous and sweat glands, as well as hair follicles. Evidently the case here described belongs to the group of simple dermoid cysts.

A survey by New[4] of 103 dermoid cysts of the head and neck stresses the rare occurrence of laryngeal cysts of this type. Actually, amongst all the published cases only Murdock Equen[5] describes a cyst similar in location and character. It was freely movable, pale pink, two cm in diameter and pedunculated; and arose from the right cricoid. It was occasionally obstructive and could not be visualized indirectly. Its removal was as uneventful as in the case here described. Its wall was of vascular connective tissue lined with epithelium.

It is the past history that makes this case rather unusual. At the age of two the patient had been treated for a tumor of the neck. Early in January 1902 the child's mother noted a swelling on the right side of the neck which had developed suddenly. Exploratory operation revealed the tumor too extensive for removal and only biopsy was done. A diagnosis of lymphosarcoma was made by Dr. W. B. Steiner. In March 1902 another physician had found a series of tumors from the clavicle to the mastoid bone. Submaxillary and axillary glands were involved. The clinical appearance was typically that of sarcoma. Treatment with x-ray and Coley serum did not help, but two weeks after the child returned home the tumors disappeared.

In April 1949 she developed a tumor in the right temporal region which was removed, and reported as epidermoid carcinoma of basal cell type. In November 1950 a total hysterectomy was performed. Leiomyomata, adenomyosis uteri, chronic cervicitis and a papillary cyst of ovary were found.

The epithelial cyst of the pyriform sinus is therefore the fourth abnormal growth in this patient. Two of them are definitely cystic, one clearly malignant, and one not definitely established. It was not possible to obtain any histological description or slides of the tumor of the neck treated 50 years ago. The patient's description and the response to treatment suggest cystic hygroma or lymphangioma, both extremely rich in fibrous cells, histologically often inseparable and both amenable to cure by x-ray or radon seeds. No doubt some sections from the first biopsy may have given the impression of malignant changes.

The ovarian cysts, two of them, removed in 1950 also showed a smooth lining and low columnar epithelium, containing gelatinous matter. Assuming that the neck tumor was originally of cystic origin, we are comparing 3 cystic growths in the same patient. Although histologically there is no common denominator for the appearance of the cyst walls, multiplicity of cysts should be noted. Whether their origin is found in an anlage or in different stimuli cannot be ascertained unless the patient should develop more cysts.

It is also of interest that two types of malignancy (lymphosarcoma and basal cell carcinoma) have been diagnosed and successfully treated in the same patient.

BRADLEY THEATRE BLDG.

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XXVI

THYROTOMY APPROACH FOR ARYTENOIDECTOMY IN BILATERAL ABDUCTOR PARALYSIS OF THE VOCAL CORDS

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Surgical procedures to provide adequate breathing space and eliminate the tracheotomy tube in bilateral abductor paralysis have concentrated principally on the arytenoid. These consist of either free mobilization of the arytenoid and lateral fixation, or removal of the arytenoid. Most procedures are extralaryngeal, and others are intralaryngea.

Baker[1] in 1916 reported a case of post-diphtheritic paralysis in which he removed a cord and the arytenoid through a thyrotomy with a good result. Hoover[2] reported submucous resection of the vocal cord and vocal process of the arytenoid. Lore[3] advised submucous resection of the vocal cord and complete removal of the arytenoid. King[4, 5] condemned removal of the vocal cord because it altered speech, and instead advocated lateral fixation of the arytenoid to the omohyoid and, later, omitted fixation to the omohyoid. Morrison[6] reported on the King technique without using the omohyoid muscle. McCall and Gardiner[7] also reported on lateral fixation of the arytenoid. Clerf[8] reports favorably on free mobilization of the arytenoid and fixation to the posterior border of the thyroid cartilage. Kelly,[9, 10] in an excellent review of the work by others advocated the extralaryngeal approach of King, but removed the arytenoid through a window in the thyroid ala. Wright[11] followed the Kelly procedure, removing the arytenoid with good results. Orton[12] used the lateral transthyroid approach of Trotter to get better access to the arytenoid for removal. Woodman[13] removed the arytenoid from behind the ala and tied the vocal process to the inferior cornu. Lynch[14] also advocates the Woodman extralaryngeal approach, because a better airway was established in his cases.

Thornell[15, 16] reported successful results by an intralaryngeal approach through the suspension laryngoscope. He dissects out the arytenoid and controls hemorrhage with the electrocautery. An acrylic obturator is inserted between the cords and removed three to four weeks postoperatively. Von Leden[17] has recently reported favorable results using the Thornell technique. DeBord reported a case of bilateral abductor paralysis for which he did a unilateral arytenoidectomy through a thyrotomy, a procedure similar to that reported in this paper.

OPERATIVE PROCEDURE

With the patient's shoulders supported by a sandbag and the head thrown back to tense the laryngeal region, sodium pentothal was given intravenously and nitrous oxide and oxygen was given through the tracheotomy tube. The skin was prepared in the usual manner. Local infiltration in a rectangular pattern of one per cent procaine with adrenaline was done to diminish the need for profound general anesthesia. A transverse incision through the previous scar or a fold was made, and the skin and superficial fascia were mobolized. The midline fascia was divided exposing the thyrohyoid membrane, thyroid cartilage and cricothyroid membrane. A No. 10 Bard Parker knife was placed in the superior thyroid notch, and the thyroid cartilage was cut in the midline. In one case (I. J.) because of ossification, a blunt-tipped shears was used to divide the laminae precisely in the midline between the anterior ends of the vocal cords. The two halves were retracted and the pharynx and trachea packed off with gauze.

The interior of the larynx was swabbed with 10% cocaine solution. An incision one cm long over the medial surface of the right arytenoid was made, exposing the cartilage. After further freeing of the arytenoid from the muscular attachments with a submucous elevator, it could be steadied with an Allis forceps. Then, by further dissection, it was disarticulated and the vocal process was freed from the cord, enabling delivery of the intact arytenoid cartilage. An electrocautery tip was inserted into the space previously occupied by the arytenoid cartilage. This was done to control bleeding and to promote lateral fixation of the vocal cord by scar tissue contracture. The packings were removed from the trachea and pharynx and the alae of the thyroid cartilage were allowed to fall together. The alae were brought into accurate apposition, and the outer perichondrium sutured with catgut. The fascia was also sutured. A small cigarette drain was inserted under the skin flap, and the skin was closed with black silk interrupted sutures.

CASE REPORTS

CASE 1.—A. N., a 58 year old white female entered a general medical ward on December 28, 1950 with labored stertorous breathing. The history stated that the attacks occurred about every two weeks, and she snored so loudly that it was difficult for others in the house to sleep. The symptoms followed a goiter operation performed a year ago. Between the attacks she was short of breath.

Physical examination and laboratory tests were not revealing. Ear, nose and throat consultation two days later revealed the cause of the distress to be bilateral abductor paralysis. The cords failed to move on deep inspiration as seen with a mirror. Direct laryngoscopy showed the cords to be adducted and the arytenoids could be moved with an instrument. A four mm bronchoscope was passed between the cords and a tracheotomy was made through the third and fourth rings with insertion of a No. 5 cannula. One week later a thyrotomy with removal of one arytenoid was carried out.

On the fourth postoperative day mirror examination showed marked edema of the right aryepiglottic fold; two days later a definite three to four mm posterior opening could be seen on inspiration with the cannula occluded. On the eleventh day this triangular opening was estimated as five mm and the tracheotomy tube was removed and the skin edges bridged. The postoperative course was uneventful. She was followed in the clinic for six months, and it was found that her voice was better than it had been preoperatively and she was able to do all her housework.

CASE 2.—I. J., a 51 year old Negress, entered the hospital with the complaint of having to breathe through a tracheotomy tube for the past nine years. A tracheotomy had been performed on the first postoperative day following thyroidectomy in 1942. Her speech was good, but was interrupted by finger occlusion of the tube for inspiration. She wished to be rid of the tracheal stoma so as to obtain employment.

Laboratory findings were within normal limits. Physical examination was normal except for the transverse scar and tracheotomy site. Indirect laryngoscopy showed both cords in the midline, and they remained together on attempted deep inspiration with the cannula occluded. Dyspnea and stridor occurred when the tracheotomy tube was blocked with the finger. On direct laryngoscopy the cords were seen to be together, and the aryteniod cartilages were freely movable on instrument palpation.

On the fourth postoperative day indirect laryngoscopy showed marked edema of the right arytenoid area. On the eigth day a posterior space between the vocal cords due to lateral position of the posterior third of the right vocal cord was noted. She was discharged and followed as an outpatient. Three weeks postoperatively indirect laryngoscopy showed a four mm gap between the cords. The tube was sealed with the obturator for 24 hours, and the patient remained comfortable. The tracheotomy stoma was closed under local anesthesia. Her voice showed no change from that preoperatively except that she commended that the volume was slightly diminished.

CASE 3.—F. P., a 33 year old Negro entered the hospital on April 17, 1951 because of hoarseness and dyspnea for two months. He had worked in a bowling alley and drank one pint of whiskey daily. For the past two months he had been confined to bed because of fatigue and shortness of breath.

Physical examination was normal except for the stridulous respiration. Indirect laryngoscopy showed both cords in the midline with slight passive movement on inspiration and expiration, allowing a two mm airway. He spoke only in a whisper. Thiamine chloride, 50 mgm, was given intramuscularly daily, on the presumption that this was a peripheral neuritis. He slept well, although his snoring at night disturbed others.

Laboratory examinations were not revealing. Neurologic consultation disclosed no other abnormalities, and a tentative diagnosis was made of toxic neuritis of unknown etiology.

One week after admission he developed a slight cold and respiration became more difficult. Mirror examination showed edema of the vocal cords, and it was deemed necessary to do a tracheotomy. A five mm bronchoscope was inserted between the cords and a tracheotomy was done through the third and fourth rings. After the tracheotomy, direct laryngoscopy showed the vocal cords in the midline, and the arytenoids were movable with an instrument. Two weeks later the larynx still showed bilateral abductor paralysis. It was recommended that the patient leave the hospital and be followed in an outpatient clinic, but he wished to be free of the tube. Therefore, a thyrotomy with removal of one arytenoid was performed.

On the twelfth postoperative day, mirror examination showed a five mm glottic chink and the tracheotomy tube was removed. He returned to work and, on several subsequent clinic visits, his voice was found to be hoarse.

CASE 4.—J. C., a 46 year old white male, was admitted on December 5, 1951 with a history of dyspnea and shortness of breath for 24 years, the onset of which he attributed to a tonsillectomy performed in 1928 while in the Navy. The subsequent loud snoring, he said, brought about his discharge from the Navy.

General physical examination was normal. Laboratory findings were not significant. On indirect laryngoscopy, the cords were seen to be fixed in the midline and failed to abduct on inspiration. On direct laryngoscopy the cords remained in the midline, although the arytenoid cartilages were freely movable on instrument palpation.

Daily administration of thiamine 50 mgm intramuscularly for one month failed to alter the clinical picture, and a tracheotomy was therefore performed followed by removal of the right arytenoid through a thyrotomy. The postoperative course was uneventful and eleven days later the tracheotomy tube was occluded. At this time there was a glottic gap of four mm, and 48 hours later the tube was removed. Three days later he was discharged and, when seen in the outpatient clinic he stated that he was able to work. His airway remained clear although his voice seemed weak.

SUMMARY

Four cases of bilateral abductor paralysis of the vocal vords are presented, which were treated by arytenoidectomy through a thyrotomy. The operations were successful in that all patients have adequate airways and useful voices.

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XXVII

ANGINOSE TYPE OF INFECTIOUS MONONUCLEOSIS REQUIRING TRACHEOTOMY

A CASE REPORT

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Of the many cases of infectious mononucleosis that are seen every year, the anginose, or pharyngeal type, is the most common. Quite often these cases are misdiagnosed, as infectious mononucleosis frequently conceals its true nature. It can masquerade as acute tonsillitis, peritonsillar abscess, acute pharyngitis, Vincent's infection, or even diphtheria. The anginose type especially is probably frequently confused with these conditions. The widespread involvement of lymph nodes in this disease may produce symptoms in the groin, abdomen, or neck. Liver and spleen involvement can simulate acute abdominal conditions, and splenic rupture can actually produce an acute surgical abdomen. Most clinicians are familiar with these commonly seen cases of infectious mononucleosis but frequently overlook the atypical forms. Joyce[1] emphasizes the importance of looking for infectious mononucleosis in any ill-defined infectious process. Though patients with the anginose type exhibit a diffuse pharyngitis and a tonsillitis, prostration is usually no more marked than in simple tonsillitis or pharyngitis, and severe respiratory difficulty due to obstruction of the pharynx or larynx is uncommon.

Exceptionally few cases with respiratory obstruction requiring tracheotomy have been reported. A review of the available literature reveals three cases,[2, 3, 4] of which one case was due to glottic edema following an injection of neoarsphenamine and not to a pathological process of the disease itself. Librach[4] in his report mentions three more cases.

The following is a case report:

A 17 year old white male was admitted to this hospital from another station with the diagnosis of acute tonsillitis. His complaints were a severe sore throat and dysphagia following the onset of fever,

chills, malaise, and generalized aching, approximately 36 hours before. Upon admission, examination revealed a moderately ill patient who was breathing audibly with his mouth open. The temperature was 100.8. The tonsils were enlarged, meeting in the midline and covered with a membranous exudate. The faucial areas including the soft palate and uvula were markedly edematous and injected. Enlarged, tender lymph nodes were palpable bilaterally in both the anterior and posterior cervical chains, the axillae, and inguinal regions. The patient's abdomen was generally tender, but the liver and spleen were not palpable. The remainder of the physical examination was essentially negative. Laryngoscopy was not attempted because of the condition of the patient's pharynx.

Laboratory studies revealed a white cell count of 17,700 with a differential of 52% neutrophils, 46% lymphocytes, and 2% monocytes. Peripheral blood smears showed a moderate lymphocytosis with numerous large forms, many with lobulated and dense nuclei and others with vacuolization of the cytoplasm. The heterophile antibody titer performed on the day after admission, was 1:224. A culture taken from the tonsils was reported as showing yeast-like organisms. Remaining laboratory studies, including red cell count, hemoglobin, urinalysis, and serology were within normal limits.

Treatment consisted of terramycin 500 mgm and cortisone 50 mgm every six hours, 10 cc of gamma globulin given intramuscularly, intravenous fluids, and symptomatic therapy. The cortisone was given for its possible effect on the edematous pharyngeal tissues. Twenty-four hours later the cortisone was discontinued. The gamma globulin was repeated on the third day.

On the day following admission, the edema of the soft palate had increased, and the patient had become cyanotic. A tracheotomy was performed, and the respiratory distress was immediately relieved, the patient's color returning to normal shortly thereafter. The fourth day after tracheotomy the patient's condition had improved to the point where decannulation was possible. Indirect laryngoscopy at this time revealed an essentially normal hypopharynx and larynx.

Ten days after operation, the tonsils had regressed to their normal size, but there were palpable nodes remaining in the cervical, axillary, and inguinal regions which persisted for another eight days. Two weeks after admission the heterophile antibody titer had decreased to 1:28. However, a peripheral smear still showed a lymphcytosis and many atypical lymphocytes with vacuolated cytoplasm. This persisted another two weeks. Five weeks after onset of the disease, the patient was considered fit for return to duty.

COMMENT

Ravenna and Snyder[5] feel that there is a type of edema of the pharynx suggestive of infectious mononucleosis. They describe a brilliant red color of the pharynx extending over the faucial arches, soft palate, and uvula; the latter appearing semitranslucent and having a pear-shape. These findings were present in over half of the cases they observed. In differentiating infectious mononucleosis from acute tonsillitis or pharyngitis, the semitranslucent character of the edema, if present, is of important diagnostic value. In our experience, we have never noted it to be present in the two latter entities.

It is important to think of infectious mononucleosis before a diagnosis of tonsillitis or pharyngitis is made, especially if lymphadenopathy is present in regions other than that of the cervical chains. A peripheral smear and heterophile titer will be confirmatory in most cases. The case presented illustrates this well.

Naturally, the question arises as to whether or not a tracheotomy was imperative and necessary. This patient had extreme air hunger, was cyanotic, and had become fatigued. Though the use of an oxygen mask improved his color, it did not alleviate his other symptoms of air hunger and this, we feel, is justification for tracheotomy. There are some who would rather defer this procedure and give the patient a chance to respond under conservative management. However, we believe that under these conditions, relieving the patient from fatigue is an indication for tracheotomy second only to the impending anoxia. It is a relatively simple and safe procedure, to which there are no contra-indications, and the resulting tracheostomy heals rapidly after decannulation.

SUMMARY

1. A case of infectious mononucleosis, anginose type, requiring tracheotomy is presented.

2. The pharyngeal findings are discussed briefly.

3. The indications for tracheotomy in this case are discussed.

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XXVIII

REMOVAL OF PLASTIC BULLET FROM TRACHEOBRONCHIAL TREE WITH A BRONCHOSCOPIC CAUTERY

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In recent years, plastics have been used in the manufacture of a wide variety of objects. When a toy plastic bullet was aspirated by a small child, it presented an interesting problem. This case is reported because of the unusual method by which the foreign body was removed.

REPORT OF A CASE

R. F., a three-year-old white boy, aspirated a toy plastic bullet on Christmas day. Immediately, an almost continuous cough developed, and he was taken to an otolaryngologist who had roentgenograms made of his chest. The films indicated partial atelectasis of the left lung, and the following day the patient was referred to one of us for treatment.

Examination revealed diminished breath sounds over the left lung with the mediastinum shifted to the left. His temperature was 102.6,° his respiration was 36.

A duplicate of the foreign body was examined. It was a toy bullet of drab-green plastic material measuring 6 mm x 13 mm (Fig. 1).

Bronchoscopy was done on December 26, 1952, without anesthesia, using a 4 x 26 Jesberg bronchoscope. The non-pointed end of the plastic bullet was readily seen impacted in the left lower lobe bronchus. There was no forceps space. Even after shrinking the bronchial mucosa with 1:6000 epinephrine solution, there was not enough space to admit the blades of any of the numerous forceps available. After working for nearly thirty minutes, we discontinued the operation.

From The Charlotte Eye, Ear and Throat Hospital.

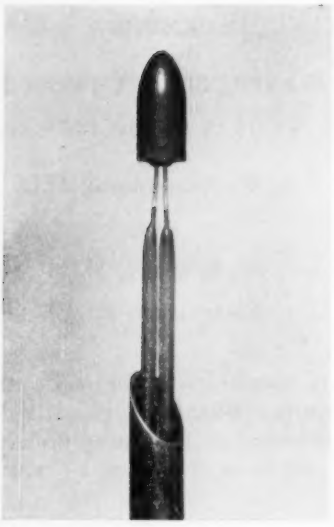

Fig. 1.—Foreign body fused to cautery tip projecting through 5 x 26 Jesberg bronchoscope

On December 27, 1952, the duplicate bullet was examined again and all forceps carefully adjusted to the width of the foreign body, prior to another attempt to remove it. At this time, it occurred to one of us that the plastic would not only melt if touched with a hot cautery tip, but that it might become adherent to the tip after it was allowed to cool. We experimented with the duplicate and found that the bullet stuck so firmly to the cautery tip that it could not be pulled off until the cautery was reheated.

A classical low tracheotomy was done, and a 4 x 26 Jesberg bronchoscope was introduced through the incision. The foreign body was identified, and again it was impossible to get forceps space, even after shrinkage with one-half cc of one-half percent neosynephrine solution. Several attempts were made, using different forceps, but none met with success. A 5 x 26 Jesberg bronchoscope was substituted. After repeated efforts and cleansing of the field, satisfactory contact was established between the foreign body and a bronchoscopic electric actual cautery tip.* The cautery was turned

*This delicate, actual cautery electrode is similar to a laryngeal electrode but measures 50 cm in length. It has been used in this hospital for several years, having been made for us by George P. Pilling and Son Company, Philadelphia, Pennsylvania.

on briefly and then allowed to cool for a few seconds. The cautery was then withdrawn with the bronchoscope, and the foreign body was firmly attached. A number 1 tracheotomy tube was placed in the trachea.

His postoperative course was uneventful, and he was decannulated and dismissed from the hospital on January 2, 1953.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, November 3, 1952

THE PRESIDENT, DR. LAWRENCE J. LAWSON, IN THE CHAIR

TRANSANTRAL EXCISION OF THE SPHENOPALATINE GANGLION AND MAXILLARY NERVE FOR FACIAL PAIN

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Over a period of years, I have been impressed with the number of patients complaining of facial pain, who have been treated by others, some with good results, others with troublesome complications and many that were unimproved. The purpose of this paper is to revive a surgical procedure and to arouse an interest in those of us who are inclined not to refer this type of patient to others.

The term facial pain, as described in the literature could be anything from a simple headache to migraine, or neuralgia, or a typical tic douloureux. No attempt will be made to discuss any of the controversial points regarding the Sluder syndrome[1] vidian neuralgia,[2] or to qualify the exhaustive study of Glaser[3] on facial neuralgia, or Fay[4] on atypical neuralgia and vascular pain, the interesting findings of McLaurin[5] regarding the reflexes caused by the fifth nerve irritation, or the Costen syndrome.[6]

Trigeminal neuralgia, which accounts for 75% of the major neuralgias of the face[7] has a characteristic symptomatology. The history is developed by the internist, otolaryngologist, dentist, neurologist, and finally the neurosurgeon, who either injects alcohol into the nerve branches or sections the sensory roots of the gassarian ganglion. The chief objection to the alcohol injection is the un-

controllability of the spread, which may destroy the function of the cranial nerve in that region. The sectioning of the sensory root produces numbness of one side of the face, tongue and homolateral eye, and occasionally persistent paresthesia, burning and itching.

Trigeminal neuralgia was first recognized by Avicenna in A. D. 1000 and described by Schlictung in 1748. Nicolies Andre in 1756 first used the term tic douloureux and in 1773 it was accurately described by Fothergill.

The pain in trigeminal neuralgia is always confined to the sensory distribution of the fifth nerve and is the most severe type of pain that can be experienced by the patient in medicine. It occurs in either sex, more on the right side than on the left, and mostly involves the second and third division of the fifth nerve. The average age of onset is around 50, but has been found in patients as young as 12 and as old as 87. These have often been seen by the dentist, have had healthy teeth removed and have usually undergone needless sinus surgery.

Trigeminal neuralgia is characterized by stabbing, flashing pains along one or more branches of the fifth nerve. The paroxysms of pain are usually started by external irritation of the involved areas such as washing the face, cleaning the teeth, chewing, swallowing, talking, yawning or exposure to drafts. Frequently there are trigger zones. The patient will protect this area by not talking, eating, laughing, or washing the face or shaving. These paroxysms may last seconds or minutes, and the patient may become excited or go into contortions, or scream for relief. These attacks may cease spontaneously. Sewall[8] and Poe,[9] have cited cases in which the gasserian ganglion has been severed with no relief. This was obtained only after the sphenopalatine ganglion had been excised. In some cases, the stripping of the sympathetic nerves from the carotid arteries failed to bring relief. The surgical attack on the sphenopalatine ganglion began with Carnochan[10] in 1858, who excised the maxillary nerve by incision through the cheek to the foramen rotundum. Vogel[11] in 1929, removed the sphenopalatine ganglion in nine living people for atrophic rhinitis. Gessenbaur, Friedlander, Koches, Lucke, Lossen, Brau, Krause and Lever, approach the pterygopalatine fossa by removal of the malar bone. In 1931, Braenckner,[12] removed the wall of the antrum through the cheek, in 1935, Auerbach,[13] resected the hard palate to gain access to the sphenopalatine ganglion. The technique employed in all the cases operated upon is as follows:

OPERATION

The usual pre-operative medication is employed and the alveolar margin and canine fossa are infiltrated with 1% novocaine with adrenalin on the side involved. The infraorbital nerve, is also infiltrated at its exit. The sphenopalatine ganglion is infiltrated with 2 cc of the solution through the large palatine foramen. The usual incision is made as in the Caldwell-Luc operation and the anterior antral wall is removed. The infraorbital nerve is isolated in the bony canal by removing the floor of the infraorbital canal with dental picks, explorers and bone forceps. The nerve is dissected from the soft tissue of the cheek to the posterior wall of the antrum and is severed at the cheek and grasped with forceps. The posterior antral wall is not thick, but gains strength from its conical form. The periosteum and posterior antral wall are next removed with the sphenoid sinus instruments. A dense layer of fascia is encountered which is excised, exposing the contents of the pterygopalatine fossa embedded in fatty tissue. The internal maxillary artery is seen pulsating in a bulging arc across the field and can be followed to the nose. If bleeding occurs here, it may be necessary to ligate the internal maxillary artery or its infraorbital branch. Extending downward and medially from the maxillary nerve near its exit from the foramen rotundum, the sphenopalatine ganglion is found. It lies outside the nose, anterior to the base of the pterygoid process and posterior to the internal maxillary artery as it approaches the sphenopalatine foramen. The ganglion is freed by careful dissection and the maxillary nerve is severed near the foramen rotundum, removing it intact with the sphenopalatine ganglion, which completes the neurotomy. Packing may be used if necessary and the usual sutures.

REPORT OF CASES

CASE 1.—C. B., a white male, 62 years of age, was first seen on January 6, 1952, complaining of severe pain in the right eye, orbit, cheek, right upper lip, and a dull ache in the right ear and right side of the neck. The pain would come on suddenly without warning. The pain was so excruciating that he could not touch the lip or nose on the right side, so that it was imposible for him to wash or shave. Because of the intensity of the pain, he occasionally had projectile vomiting. During this time, he was being treated for sinus trouble localized to the right side. In the previous five years, he had suffered two attacks of coronary thrombosis. He had consulted his dentist on numerous occasions, who could find nothing wrong with his teeth. In desperation, he finally pulled two suspicious teeth with no relief, and was advised to change climates. He next visited two otolaryngologists, who gave him routine treatment, with no relief. As a last resort he visited a naturopath and was again advised to go to live in another climate. On his first visit the right sphenopalatine ganglion was anesthetized with 10% cocaine, which relieved all of the pain in the right eye, ear and nose, and reduced the intensity of the trigger action on the lip and nose approximately 50%. On his next visit two days later, the patient had a recurrence of symptoms and the same procedure was repeated with relief as described above. Because of

the intense pain and the possibility of precipitating another coronary attack, the patient was hospitalized on January 19, 1952, and completely examined by his cardiologist, in preparation for surgery. While in the hospital the right sphenopalatine ganglion was injected with 1% novacaine, which gave him complete relief immediately, but three hours later, the pain returned and was so intense that a half grain of morphine intravenously produced no relief. It was impossible to restrain the patient in bed because of the pain. On January 20, 1952, the maxillary nerve and sphenopalatine ganglion were removed with complete relief of symptoms. The patient was last seen on October 6, 1952, and now has no pain. He is eating, swallowing, laughing, and chewing and has gained twenty pounds which he had lost.

The case represents the typical patient on whom the neurosurgeon sections the gasserian ganglion or injects the affected division of the fifth nerve. Love[14] describes a new treatment for trigeminal neuralgia of decompressing the gasserian ganglion and its posterior root. The otolaryngologist obtains the same result with a minor surgical procedure.

Case 2.—D. L., a white, married female aged 32, was first seen on January, 1948, referred by an ophthalmologist and a family physician. At the initial examination she complained of pain in the left eye, cheek, teeth and ear, which radiated down the neck. With this she would have nausea and vomiting. The ear, nose and throat examination at the time was essentially negative. The left sphenopalatine ganglion was cocainized with complete relief of symptoms. For the next two years this patient was seen at various times with the above complaints, and in addition, stated that frequently her left eye was swollen shut, and the entire left side of the face would become edematous. The pain was so severe that aspirin and narcotics were unable to control it. On January, 1950, she was examined in the Department of Neurology at Research Hospital and was subsequently transferred to the psychiatric department, for therapy, without improvement. She returned on January, 1951, with the same complaints. In February, 1952, she was hospitalized at the South Chicago Hospital with pain in the left side of the head, face, teeth, neck, shoulder, left arm and back. A neurologist was called in consultation and made a diagnosis of neurosis. During her stay in the hospital all laboratory findings were negative and her symptoms were controlled by an injection of the left sphenopalatine ganglion, which relieved her for three days. Then the pain returned with greater intensity. In June, 1952, the sphenopalatine ganglion and maxillary nerve were removed, with complete relief of symptoms.

This type of case taxes the diagnostic ability of all branches of the profession, the patient submits willingly to all types of medical and surgical therapy, and finally emerges with his neuralgia still intact.

Case 3.—T. L., a white, female nurse, age 39, was first seen on January 8, 1952. She first complained of headaches on the left side of the head and a dull ache in the left ear. The left eye and cheek would become edematous, and there was diminished hearing in the left ear. The pain would radiate down the neck to the left shoulder. Coainization of the left sphenopalatine ganglion produced relief. The pain continued to become more intense, swelling persisted. X-rays of the teeth were negative, and the sinuses showed some clouding of the left maxilla, which was irrigated with no relief of symptoms. In February, the pain in the left cheek, eye and head became very severe so that this patient was unable to work as night supervisor. Opiates gave her no relief. On February 15, 1952, the left sphenopalatine ganglion was injected with 1% novocaine, which gave her

complete relief of symptoms for three days, but the pain of the left side of the head returned with greater intensity. In addition, there was blurring of vision of the left eye.

All this time the patient was under demerol and on March, 1952, she complained of diplopia, in the left eye and numbness of the left side of the face and the entire left arm. She developed a staggering gait and was unable to judge distances. Pain in the left ear and upper teeth persisted, so that the patient was hospitalized and examined by a neurosurgeon. The left sphenopalatine ganglion was again injected with complete relief of symptoms for two days.

In April, 1952, with the pain still persistent, the patient became drowsy and was again hospitalized. A spinal puncture was done by the neuro-surgeon. The headache became intense and there was numbness of the left side of the face and blurring of the left eye. For the first time on April, 1952, she developed a trigger zone on the left side of the nose. An encephalogram was made by the neurosurgeon to rule out a central lesion. During the process of doing this, the patient developed sharp pain in the left side of the chest with dyspnea and cyanosis. A presumptive diagnosis of pulmonary embolism was made. The patient was placed in an oxygen tent. Aminophyllin, heparin, and dicumerol were given. On the second day, while in the oxygen tent, she developed a pumping sensation in the upper left first molar, which lasted approximately ten hours, and while convalescing at home one week later, she was awakened in the middle of the night with a tooth ache in that tooth. A dentist refused to extract the tooth. I insisted the tooth be extracted. On one root was found a localized abscess. After the extraction, the hearing seemed to improve and all the pains on the left side of the face, numbness, diplopia, and blurring of vision disappeared.

Although this patient had been scheduled for transantral excision of the sphenopalatine ganglion and maxillary nerve, the neurosurgeon was suspicious of a central lesion, which subsequently was disproven. Fortunately, a tooth became the trigger zone, but the maxillary neurotomy with removal of the sphenopalatine ganglion would have produced the same result.

SUMMARY AND CONCLUSION

In reviewing the literature, it is apparent that the symptoms produced by the gasserian ganglion may be difficult to differentiate from the sphenopalatine ganglion complex. It appears impossible to define within limits the major neuralgias. Claims are made that the only cure for the gasserian ganglion complex is the division of the nerve, which is not always true. The question often arises which of these to attack, the sphenopalatine ganglion or the gasserian ganglion. Frequently, the neurologist cannot make a decision, as has been demonstrated by Sewall and Poe. There have been instances in which resection of the gasserian ganglion has been unsuccessful. When there is a question or a doubt, the sphenopalatine ganglion and the maxillary nerve should be excised first, because the gasserian ganglion, which is a major ganglion can always be resected later.

The removal of a sphenopalatine ganglion should be considered for all patients with pain referred to the eye, orbit, temple, cheek, neck or shoulder, even when it goes down the arm to the fingertips.

If there are any symptoms referred to the teeth, no matter how minimal they appear, the maxillary nerve should also be removed. Some patients also complain of pain in the back extending down the leg to the foot, with tingling and quivering of the toes which has been ascribed to the sphenopalatine ganglion (McLaurin). One cannot always be certain that there exists a maxillary neuralgia when facial pain is present. Frequently, the ophthalmic and mandibular divisions are involved as well as fibers of the sympathetic nerves. The question arises whether the sphenopalatine ganglion should be removed by itself or along with the maxillary nerve. If there is any doubt, and even in the absence of trigger zones, I always remove the maxillary nerve and the sphenopalatine ganglion.

I believe that the rhinologist is best qualified to cope with this problem both medically and surgically. The patient with chronic recurrent head pain becomes a responsibility of the otolaryngologist.

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DISCUSSION

DR. MILTON TINSLEY: I have enjoyed Dr. Morse's paper, but I wish to discuss primarily trigeminal neuralgia. I shall not discuss any phase of the sphenopalatine ganglion syndrome; whether it is possible to inject the ganglion or whether, in injecting the ganglion, one does also inject the maxillary nerve. Nor shall I argue who is the culprit when the maxillary nerve is removed with the ganglion. The diagnosis of trigeminal neuralgia is not difficult. There is nothing that can be mistaken for trigeminal neuralgia. It is confined to the distribution of the sensory division of the fifth nerve and there is no overlap of pain. The pain is paroxysmal. The treatment is either injection of the nerve or section of the sensory root, with relief.

In the first case presented, if we disregard the pain in the neck and ears we may consider that Dr. Morse was dealing with typical trigeminal neuralgia; that is, if there was no pain between paroxysms. The relief upon section of the maxillary nerve has persisted for more than eight months. Shall we await regeneration of the nerve before judgment as to the efficacy of the procedure? We know that the fifth nerve cannot be killed in its peripheral distribution. One can avulse it, one can inject it, one can burn it, one can put objects into the foramina, and it always regenerates and the pain recurs. We should wait until this nerve has had a chance to regenerate, which may take a year or two years.

In the second and third cases, certainly neither showed trigeminal neuralgia. In the former, the distribution of pain was not typical. In the latter, as implied and presented, the symptoms were entirely on an infectious basis, producing intracranial and extracranial signs and symptoms. I am at a loss to understand why Dr. Morse believes that removal of the sphenopalatine ganglion would have cured the abscessed tooth.

In speaking of trigeminal neuralgia we should refer to a definite condition and not speak of overlapping symptoms and signs. So far as the statement that recurrent head pain should be in the realm of the otolaryngologist, Dr. Lederer reminded me of a patient we had, who had an acoustic neurinoma, who was treated for sphenopalatine neuralgia by sphenopalatine injection. Recurrent pain in the head may be due to brain tumor or to various other diseases. Also, in the so-called Sluder's neuralgia or sphenopalatine neuralgia, the distribution of pain has been found frequently to be associated with a herniated intravertebral disc in the cervical region.

The only permanently effective treatment of trigeminal neuralgia is destruction of the preganglionic fibers of the sensory root by alcohol or by section of the sensory root, or by section of the spinal fifth tract in the medulla, a medullary tractotomy. The work of Dr. Love on decompression of the fifth nerve has not been repeated by anybody else. Many times men have gone in and have been unable to section or expose the fifth nerve due to bleeding, and have had to terminate the operation, and the patients have been relieved of trigeminal neuralgia for awhile. Trigeminal neuralgia is a condition which has spontaneous remissions and exacerbations and any medical treatment advocated must be considered on the basis that possibly the treatment happened to coincide with a remission.

DR. ROBERT LEWY: At the risk of superarrogation, in speaking after a neurosurgeon, I should like to make some comments as an otolaryngologist. We should remember that Sluder's work was by no means always agreed to by others. Vail objected to it strenuously on the basis of what he thought was the vidian nerve being involved; Higbee had some objection to it, as did Proetz in discussing Higbee's paper; Larsell indicated in some work which he had done that there were fibers of the maxillary division of the fifth nerve which went through the sphenopalatine ganglion.

I must say also that I am not impressed in the clinical history that these are necessarily typical cases of sphenopalatine neuralgia. Further, when both the sphenopalatine ganglion and the maxillary nerve are avulsed surgically, I do not know how one can decide exactly which element is giving the effect described. I recognize that there is no substitute for results, and am pleased that Dr. Morse has apparently obtained these results, but I do not think there has been substantive proof offered, anatomically, that the sphenopalatine ganglion is specifically involved, except insofar as the effect of cocainization or novocaine on the sphenopalatine ganglion apparently gave relief. As Dr. Tinsley pointed out, the sphenopalatine ganglion is very close to the maxillary division of the fifth nerve, and injection of one may involve the other. Another element which I think confuses the picture to some degree and makes the ultimate reasoning difficult, is the work done by Lytell on the anterior ethmoidal nerves some years ago. You may recall that he did cadaver dissection first, to actually cut the ethmoidal nerves, and proved that by cutting the ethmoidal nerve he could succeed in correcting pain located in some of the regions described by Dr. Morse. This concept is, of course, more or less academic. As I said, what counts is the results, but I cannot be convinced on the basis of the material offered that the results are necessarily due to any work on the sphenopalatine gang-

lion, and it would be quite possible that the same results might have been obtained had the sphenopalatine ganglion been let alone and the maxillary division of the fifth nerve been sectioned.

Dr. Leo M. Morse (closing): When I presented these patients before the University of Chicago group this afternoon, I mentioned that I came for constructive criticism, that I was not prepared to enter into the academic reasons, and that all I had was the practical results. I have been accused of trying to create or revive an operation because of our so-called dying specialty; maybe that is so.

I am not in disagreement with Dr. Tinsley. If we break down this classification and speak of the patient with the typical trigger zone, the otolaryngologist is offering a simple minor procedure in a field in which he is accustomed to work; he knows the anatomy and can perform the operation in a short time. Whether or not he is doing the right thing from the standpoint of research and anatomy, and neurophysiology—I cannot say.

Dr. Tinsley thought the third case was on an infectious basis. All these patients had had complete physical examinations from every standpoint, and Dr. Rasmussen mentioned at the time that this patient might have had a mild encephalitis. She had had two lumbar punctures and all findings were negative. Whether or not to remove only the sphenopalatine ganglion or the maxillary nerve I do not know. Whether or not the abscessed teeth remaining should be left in after the symptoms are gone I do not know. At least in my practice I get the type of patient nobody wants to take care of. They have made the rounds, have seen the neurologist, the psychiatrist and, incidentally, the third patient, who was a nurse, actually thought she was crazy and went to a psychiatrist. Fortunately, she developed a trigger zone in the tooth. Are we justified in allowing a patient to go to such lengths when this simple procedure would take care of the situation? I saw her frequently enough to be aware of the pain she suffered. I do not know what will result from the abscessed tooth that remained in her mouth, but we did take care of the pain. Incidentally, she had a 25 db loss of hearing when I first saw her, and following removal of the tooth her hearing was restored, as she tells it. I have not repeated her audiogram recently because we are giving her a chance to make a recovery.

VOICE REHABILITATION IN THE ABSENCE OF THE LARYNX, LOWER PHARYNX, AND CERVICAL ESOPHAGUS

EDWARD B. WEISMAN, M.D.
CHICAGO, ILLINOIS

With the greater trend toward radical neck surgery, the loss of tissues requires new means toward the patient's rehabilitation. My experience with a patient who had undergone total laryngectomy, partial pharyngectomy, cervical esophagectomy and right radical neck resection for an epidermoid carcinoma of the right hypopharynx which had spread to the larynx, cervical esophagus and lymph nodes, presented a problem in communication (Fig. 1). This patient was unable to write.

Although restoration of the pharyngo-esophageal lumen might be started as early as six weeks, in the hands of some surgeons, this is not always desirable or possible. The need for prolonged observation after radical surgery, the use of radiation, poor wound healing or delayed tube flap techniques are some of the deterrants to early closure.

In caring for the patient with pharyngeal, esophageal, and tracheal stoma, one must deal with (1) Loss of conventional facilities for voice restoration as exist for the laryngectomized patient, (2) Undesirable loss of salivary secretions, and (3) Unpleasant drainage from the pharyngeal stoma to the neck.

For these purposes, I have devised the following adaptations for the electrolarynx (figure 2) to meet the new situation:

(1) A latex prosthesis to seal the hypopharynx, thereby creating the necessary closed chamber and permitting the lips, tongue, palate and resonators to perform their normal functions in the voice production, as well as permitting head and neck motion.

(2) A conducting tube to allow the sound to reach the voice-producing area (and to permit operating the apparatus from the patient's side).

Fig. 1.—Patient postoperatively.

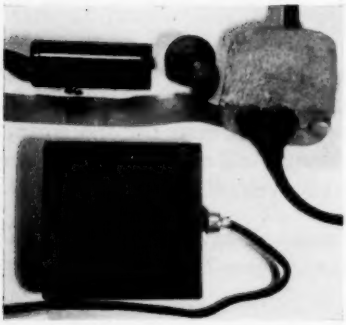

Fig. 2.—Prosthesis and electrolarynx. A. Electrolarynx with adaptor head. B. Conducting tube. C. Latex prosthesis held in place by Davol No. 262 cement and strap. Prosthesis has patent bone buttons for sound tube and for drainage tube. Receptacle section fits into neck to drain pharynx.

Fig. 3.—Patient with prosthesis applied to neck and electrolarynx and battery in case on his belt.

(3) An adaptor head for the electrolarynx to allow simple union with the conducting tube.

(4) A metal diaphragm for the adaptor head to create a higher pitched sound as required by this modification. The vibrator may also have it's tone producing qualities altered as needed.

(5) A drainage tube allowing the secretions to enter the esophagus thereby limiting the loss of saliva and improved local hygiene.

(6) The use of a surgical appliance cement for application to the prosthesis in addition to the support of the latex neck strap.

(7) A case to contain the battery and vibrator which is hung on the belt.

The rubber cement is applied to the clean, dry neck as well as to the prosthesis. After holding the appliance in place for one minute, the strap is applied to complete the fitting.

The drainage tube is inserted into the esophagus and the conducting tube is attached to the bone button in the appliance.

The battery and vibrator may be worn on the patient's belt and handled with ease and comfort. A daily morning change and cleaning is adequate.

The patient in figure three has comfortably worn his appliance and enjoyed a happier postoperative period, prior to plastic reconstruction.

SUMMARY

A modified electrolarynx was devised to be used in the case of a patient with a partial pharyngectomy, cervical esophagectomy, and total laryngectomy. This instrument is of value during the interim between radical surgery and reconstructive surgery in giving the patient a means of communication.

904 West Adams Street.

DISCUSSION

Dr. William F. Waldrop: Those of us working in the field of rehabilitation of such cases have been trying for years to find various means that would better answer the problem than the two devices usually used; the electrolarynx, which was modified in this case, and the old Western Electric artificial larynx.

We have had no such cases in our rehabilitation service, but recently Duncan Scott, the audiologist working with the speech therapist at Billings Hospital, asked if I would loan him the devices we have. We have the electrolarynx and the Western Electric artificial larynx. I asked that he return them in a few days because we like to show them to laryngectomized patients. Fortunately most of these patients who have failed with the esophageal method have bought one or the other of these devices and, shortly afterward, have learn the other method, so we had several on hand. He did return one that I loaned him but said that the patient refused to part with the other, and that they would have to buy it from us. This is evidence of the dependence of the patient on speech and, in such a case, one must resort to the device.

Most of the modifications we have tried to make have not been sufficiently adaptable to make them worth while. I hope that from this modification something will be developed which will be more flexible in its pitch range. In our experience neither the electrolarynx nor the Western Electric larynx have enough variance in pitch to be very practical. There are one or two other instruments somewhat similar to the artificial larynx with a reed which is a little more flexible, and we have recordings on persons who have obtained, in addition to the voice inflections in intensity obtained with these instruments, a certain amount of variation in pitch, so that there is

actually a pitch inflection. I would like to see such an instrument tried in a similar adaptation. This, I understand, was more a temporary arrangement to serve between the two surgical procedures, and I would certainly be interested to learn more about it. This case presented a problem which they did not have in the case at Billings Hospital, and has been solved in a very practical manner.

DR. B. J. SOBOROFF: Dr. Weisman is to be congratulated in handling so ably the problem we had at the Illinois Eye and Ear Infirmary. It was not only that of providing a voice for a patient who was unable to communicate because he could not write, who had had his larynx and pharynx removed, but also the problem of handling a stoma in the floor of the mouth which is automatically created when a laryngopharyngectomy is performed. There is little information available as to how such a double problem could be met. Other men, in such cases, have provided plastic tubes which fit into the floor of the mouth and lead into the esophageal stoma to collect secretions and provide a solution to that part of the problem, but of course that does not provide the ability to speak.

This device has not only sealed off the lower portion of the large stoma, but also provides a means of bringing the secretions down from the mouth and the upper portion of the pharynx into the esophageal stoma. It might be said that this is only a temporary measure, yet with the reports that have come from the Memorial Clinic as to the improved results in lesions of the hypopharynx with surgical procedures, with the necessity for more extensive surgery in order to obtain these improved results (in this case a laryngopharyngectomy with right radical neck dissection) it is necessary to provide early rehabilitation, and the problem then arises as to reconstruction of the pharynx. This will be the next step, but it may be months or a year before the entire hypopharynx and pharyngeal area can be reconstructed with two pedicle flaps from the chest, so it is very important that something of this sort be used in the interim. Dr. Weisman has produced a practical solution to this difficult problem.

DR. A. H. ANDREWS: We have been endeavoring for some time to solve some of the energy problems in connection with the artificial production of sound. Dr. Weisman's work suggests the application of the electrolarynx and, if successful, this will constitute a more simple solution than has been provided by our acoustical engineer.

DR. EDWARD B. WEISMAN (closing): Apropos of Dr. Waldrop's inquiry, the problem we are dealing with, as Dr. Soboroff verified, is that ordinarily the laryngectomized patient does not have the

problem of a pharyngeal stoma; there is continuity between the pharynx and the esophagus. This patient had surgery which was much more extensive. I inquired about devising a more compact electrical apparatus, and the cost of engineering something electronic was about $3,000; so I decided that this would be a little cheaper although somewhat clumsier.

Speech, Hearing and Language Disorders: A Frame of Reference For Research and Therapy

HELMER R. MYKLEBUST, M.D.

In the past disorders of communication have been considered mainly as disorders of speech. We now recognize that ability to communicate is more than ability to speak and, with this awareness has come a greater understanding of language and language disorders. Basically, study of the communication process entails study of language. For this purpose language can be divided into three areas.

(1) Expressive language. This is the ability to use language in speaking and writing.

(2) Receptive language. This is ability to understand what other people say (hearing) and the ability to understand what has been written (reading).

(3) Inner language. This is the language one uses to communicate with himself.

Disorders of language can occur in any of these three areas or in various combinations of them. Furthermore, there is a reciprocal relationship between these language functions. In order to have expressive language (speech) one must first be able to hear. There is a reciprocal relationship between hearing and speaking defects. Furthermore, the individual cannot understand what other people say, nor can he speak, until he has acquired a minimum of inner language.

We are gradually learning more about the process of language acquisition. We are now aware that there are two basic avenues through which the child learns language. These are the sensory avenues and what we have termed the psychologic avenues. It is possible for a child to have normal sensory avenues but not acquire language normally because of marked emotional difficulty, which

blocks the psychologic processes of language acquisition. In order for language to be acquired normally it is necessary for both the sensory and psychologic avenues to be working harmoniously.

Language disorders may be classified under symbolic language disorders, which are the aphasias; receptive language disorders, which derive from disturbances of the sensory processes, including both hearing and vision; (these receptive disturbances may be classified into central, peripheral and psychologic); expressive language disorders, which include dysarthrias, voice problems, delayed speech, stuttering, mutism, etc.; these, too, may be classified under central, peripheral or psychologic disorders.

Careful study and classification of the language disturbances will lead to better understanding of one of man's basic characteristics —that of ability to function symbolically. The otologist has a prominent part in this study because he is dealing with *hearing*, one of the basic senses affecting symbolic functioning in man.

DISCUSSION

DR. RICHARD E. MARCUS: It is extremely difficult to discuss the whole area of hearing, speech and language disorders, in the time Dr. Myklebust had at his disposal. It is even more difficult to get into classification of aphasias, especially in children, because at that point the controversy becomes almost insoluble.

We have seen a number of children of the types Dr. Myklebust has described. We have seen them when the parent brings the child in because at the age of two he does not speak, thinking that this may be due to the tonsils being diseased or enlarged. Where can one begin to make the differential diagnosis? It would have to start in the history obtained from the parents, especially as it relates to any changes that might occur in intrauterine life; it would have to include such possibilities as birth damages, anoxia, and anything that might demonstrate brain damage. It would then step to an evaluation of the child himself, and here is where Dr. Myklebust has tried to tell you that one of the ways in which you can approach such a child is by observing his behavior. Children who have brain damage, for instance, and who do not speak, behave in a different way from children who have a peripheral deafness and do not speak.

In addition, we have now available such things as electroencephalography, with which we feel that we can demonstrate not only whether there is or is not hearing, but also whether there is or is not brain damage. (Some of Dr. Myklebust's classifications may gain added validity as the evidence gained from electroencephalography is correlated with clinical observation.)

The field of communications disorders is one in which we can all co-operate to mutual advantage. As otolaryngologists we are in it and, I believe, to stay. We need to know the kinds of basic sciences, as Dr. Myklebust pointed out, that apply to disorders of communication, not necessarily histology or biochemistry or physiology, but rather the anatomy and function of human behavior. We are on the periphery of these areas, but we need to gain more specific knowledge in the field of human behavior especially as it relates to the rehabilitation of the patient with a speech or hearing disorder.

Dr. Myklebust's introduction to speech, hearing and language disorders is very much to the point. I should like to make one further comment. He referred to so-called "tone" deafness in children. We have seen such children and, after rather careful investigation, we find that the child originally believed to have tone deafness either was afraid of the audiometer in many instances, and did not respond until the fear had been overcome, or could not understand the kind of response that was required, and consistently waited until the volume was turned up to 50 or 60 decibels. Often such a response was maintained through many tests until we could explain that the response was expected at a lower volume. We found too that the hearing for speech for these children was perfectly normal. I do not believe that a special category is as yet necessary for that kind of child.

Dr Thomas C. Galloway: Years ago this subject seemed very simple. We knew, for example, that when we had a left temporosphenoidal lobe abscess in the area of the posterior half of the first central convolution, we interrupted connections to a sort of telephone exchange and got aphasia. It was simple, but the trouble was that we could do little about it.

One might get the impression from Dr. Myklebust's scholarly presentation that this is an abstract and impractical approach to the problems of speech. I have seen him work with these aphasic children, and one could not tell superficially whether they were imbeciles or whether they could not hear. They apparently had no contact with the realities of life, they had no opportunity for education, they were shut off from the world. By practical application of the principles he has enunciated he has taken some of those children who had been passed as very subnormal children and, in a few months in some instances, he has brought them up to a normal level of social behavior and education. This is not theoretical, it is a practical approach to the problem of speech. It depends, however, on a search-

ing analysis of many factors which we have taken for granted, and it is something we must appreciate if we are going to get results with these children who otherwise might be tremendously handicapped, but need not be.

Meeting of Monday, December 1, 1952

THE PRESIDENT, DR. LAWRENCE J. LAWSON, IN THE CHAIR

Mucocele of the Frontal Sinus

S. M. MORWITZ, M.D.

This case is presented from two standpoints: That of pathogenesis and that of surgical procedure.

T. H., a male, aged 28 when first examined in August, 1942, gave a history of two right external frontal sinus operations for mucocele and removal of the left lacrymal sac between five to seven years ago. He suffers from recurrent sinus infections. Recently there appeared a swelling in the right inner orbital region with the right eyeball pushed downward and outward. A soft protruding putty-like mass was palpable at the floor of the right frontal sinus, the eyeball was displaced downward and outward with diplopia present on looking to the extreme left. X-ray of the sinuses indicated a round mass in the right frontal sinus extending beyond the midline and through the floor of the sinus, with haziness of the left frontal sinus.

On September 14, 1942, under local anesthesia, the right frontal sinus was explored via external approach. The incision followed the existing curvilinear scar along the eyebrow and along the right border of the nose. An enormous mucocele was encountered, a section of the inferior wall of the sinus was gone, the midseptum was perforated and the floor of the sinus was devoid of mucosa, possibly from previous operations. Recovery was uneventful and the eyeball receded. Thereafter there were recurrences of short duration several times a year, with headache, prominence and recession of the right eyeball. Following each attack the Van Alyea trocar and Ritter sound readily entered the right nasofrontal duct.

On April 8, 1949, another operation was performed because a large mucocele again presented in the right frontal sinus, with marked

displacement of the eyeball and diplopia. Under intravenous pentothal the approach into the sinus was through the present scar and a large mucocele was removed. Most of the inferior wall of the sinus was gone, and most of the midseptum between the frontals. A deviated portion of the nasal septum superiorly was submucously resected, and a rubber tube was kept in the nasofrontal duct for ten days. Uneventful recovery with marked recession of the eyeball ensued. Large Ritter sounds easily entered the right frontal sinus. He still continued to have recurrent espisodes of right frontal sinusitis. In November, 1951, two and one-half years after the second operation (the fourth operation on that sinus) 2 cc of thick mucus was aspirated from a slight bulge below the right eyebrow. There was an apparent tendency toward recurrence of another mucocele. To date it has not reached large proportion because there appears to be some drainage into the nose.

At the last examination on December 17, 1951, there was no prominence in the right inner orbital angle, no diplopia, some displacement of the eyeball, no fusion. Vision was 20/40 R. E. 20/30 L. E.; with corrective lenses vision was 20/25 in each eye. Further surgery is still a possibility.

In appraisal of this case, the following conclusion become suggestive:

1. The recurrent nasal accessory sinus infection was a factor in causing the mucocele to recur in spite of a patent duct.

2. Complete removal of the sac and lining membrane of the sinus did not prevent recurrence of a mucocele.

3. Four external frontal sinus operations failed to produce a complete cure.

4. Should it become necessary to again evacuate a progressing mucocele, the Goodyear intranasal operation might now adequately provide sufficient drainage into the nose and perhaps circumvent future cystic formation in this sinus.

DISCUSSION

Dr. Jack Allan Weiss: Removal of the mucosa is a matter of judgment, and considering that the mucosa is frequently thinned and inoffensive it can and should be left. Areas of increased hypertrophy, when found, should be removed. The intranasal operation, while feasible, is certainly not applicable to the majority of cases. The maintenance of the postoperative connection between the frontal sinus and the nose, which is so essential, could be discussed at great

length. Suffice it to say that in surgery of the mucocele it seems to present somewhat less of a problem than in chronic suppurative sinusitis.

Another point is the exposure of the dura and, the possibility of intracranial complications, if a pyocele occurs. There is a variation in the incidence of dural exposure, not related to the size of the mucocele.

In a case of mine, seen in 1941 the mucocele measured 10 x 8.5 x 6 centimeters. The entire anterior wall and the entire floor of the frontal sinus were absent, and there was considerable extension backward into the orbital plate of the frontal bone. Despite all this there was no exposure of the dura, in contrast to another of my cases in which there was a small external swelling and a rather large exposure of the dura. Removal was done through the external approach, leaving a considerable defect which it was intended to repair by plastic reconstruction. However, the patient died a few months subsequent to operation, following a cholecystectomy.

The roentgenologic findings are variable depending on the size of the mucocele, the direction of its extent, the degree of erosion of the bony walls, the amount of mucosal hypertrophy, and the viscidity of the fluid content. Usually there is increased opacity of the sinus with some increase in its gross outline and smoothing of the customary scalloped border. The septa disappear. The orbital roof and anterior sinus wall may be defective or absent. There may be marginal density due to irritative osteogenesis. Paradoxically, there may be increased radiolucency with a mucocele despite the fluid content and even the possibly thickened mucosa, due to the fact that thinning or erosion of the bony anterior wall more than compensates for the amount of density of the contents and the thickened mucosa.

The sinus mucosa was thin except for the anterior wall which was about 9 mm in thickness; that portion was removed. The epithelium of the hypertrophied non-inflammatory mucosa is variable, either ciliated columnar or pseudo-stratified in areas. There was thick new connective tissue stroma with a considerable amount of new capillary formation and a scattering infiltration of cells of chronic inflammation, plasma cells and lymphocytes as well as red blood cells and fibroblasts. An occasional mucous gland was present.

DR. GEORGE H. WOODRUFF: In a large mucocele such as has been illustrated, it seems to me we should aim at obliteration of the sinus if possible; that is about the only way of obtaining a reasonably sure cure. In the smaller types, if one is able to construct a patent

duct that will stay patent, that may well suffice, but in the extremely large ones all the mucosa should be removed and enough bone to allow the sinus to become obliterated. In many cases this will cause far less deformity than anticipated.

Continuously Open Eustachian Tube with Malingering

GEORGE H. WOODRUFF, M.D.

The patient was a 44 year old, well-nourished robust male. The condition followed a fairly extensive first degree burn of the left side of the body, face and external ear. Both ear drums were normal except for slight atrophic changes. The symptoms were rumbling noise in the left ear, headache and deafness. The audiometer showed nearly normal hearing in the right and almost total deafness of the left ear.

After several examinations the left eustachian tube was found to be abnormally patulous. This was corrected by two insufflations of salicylic acid and boric acid powder. The tinnitus ceased entirely but there was no improvement in the almost total hearing loss in the left ear. After various tests were performed a diagnosis of malingering was made. This was an industrial case and true complete deafness would have been compensible. In conversation with the patient, he was told: "The examinations we have made and the tests performed show that you should hear better than you do." He said nothing at the time, but on his next visit the hearing was definitely better and the improvement continued steadily until his discharge two weeks later, when the audiometer showed the hearing to be approximately equal in the two ears at the level of twenty decibels in the speech range. The whispered voice was heard at thirty feet in each ear.

It was felt that the words used in telling the patient the results of the hearing tests were important. He was never told that he was malingering, in fact, it was never even hinted at. He was merely told that the tests showed he should hear better than he did, and he apparently took the hint.

In reply to the question as to whether the tinnitus subsided, when the tube was closed the tinnitus did subside, but the hearing did not improve. The typical history in most cases is of a roaring tinnitus. If questioned the patient will say he can hear his own breathing; the roaring tinnitus is in time with respiration. Many

patients breathe through the mouth rather than the nose because this lessens the tinnitus. The tube can be closed temporarily if the patient lies down or bends over.

Pulmonary Hemorrhage and Poliomyelitis

ROBERT LEWY, M.D.

A 20 year old white female suffered from acute poliomyelitis of the bulbar and spinal type. During the course of the disease her condition required a tracheotomy. This afforded relief and the patient proceeded to improve in her general condition. However, she developed severe hemorrhages which came from the lung and were aspirated through the tracheotomy opening. Bronchoscopic study of the pulmonary bronchial tree was done but no specific bleeding areas were found, and none of importance were found around the stoma. Despite bronchoscopy and all known general methods of control the patient bled repeatedly and finally expired.

At autopsy no explanation was found for the bleeding. The cause remains unknown, but it is suggested that pulmonary bleeding during the course of the disease, like gastrointestinal bleeding, remains a grave prognostic sign.

DISCUSSION

DR. THOMAS C. GALLOWAY: In my experience hemorrhage from the respiratory tract has been very rare in poliomyelitis. In this case there may have been some obstruction with increase of negative intrathoracic pressure and cupping action on the capillaries. That would be the simplest explanation. However, two years ago we had many cases in which, on passing a stomach tube, we aspirated blood from the stomach. We have had in three cases of poliomyelitis what appeared to be Cushing ulcer, which is supposed to be associated with a lesion in the hypothalamic area. One patient had a perforation of the stomach and the diaphragm, peritonitis and empyema. There have been two others with perforations in the esophagus.

DR. ROBERT LEWY: In reply to a question as to whether the administration of tryptar had any relation to the hemorrhage, I might say that as soon as the hemorrhage began the tryptar was stopped immediately, because while this tryptar problem was being studied it was thought that possibly it could contribute to hemorrhage. It was used in a low dilution on the series of cases. This was

the only case in which there was a possible connection between the tryptar and the hemorrhage and we do not think that there was a connection. In higher concentrations it is rather irritating.

We feel confident that in this case the bleeding was pulmonary rather than gastrointestinal, because it came directly out of the tracheal stoma and it was fresh red blood. There is some virtue in the prevention of crusting and other things which occur in the blocking of bronchi in tracheal operations of various types as, for example, in tracheobronchitis.

Otogenic Brain Abscess

PIERCE W. THEOBALD, M.D.

A 35 year old white male had a history of a perforated ear drum on the right side since early childhood, with occasional episodes of foul-smelling discharge from the ear but no other associated symptoms such as headache or vertigo. Because of his religious beliefs he had not sought medical care until on February 17, 1952, he saw a doctor because of gradual onset of pain in the right ear, mastoid and neck, with elevated temperature. Several penicillin injections were given without relief. Three days later the ear began to discharge, about normal in amount and character as compared to previous episodes. He then sought aid from chiropractic manipulations but these, too, failed to relieve his symptoms. On March 2, 1952, fifteen days after the onset of pain in the ear, he became stuporous and was admitted to the neurosurgical service of St. Luke's Hospital on March 3, with a temperature of 99.°

Examination revealed bilateral papilledema of two diopters with perivascular hemorrhages. The right pupil was large and did not react to light or accommodation. There was a third nerve paralysis on the right with ptosis, and moderate central paresis of the seventh nerve on the left.

The left tympanic membrane was normal. The right external auditory canal was filled with thin purulent material. The large attic fistula was filled with granulation tissue and cholesteatoma. The Weber test lateralized to the right. The Rinne test was negative on the right and positive on the left. There was slight left hemiparesis and hemihyperesthesia. Deep reflexes on the left were slightly hyperactive; superficial reflexes were hypo-active. Bilateral Babinski reflexes were present.

A diagnosis of right temporoparietal lobe abscess was made. The day following admission, the abscess was located by an approach through the temporoparietal region and about 30 cc of foul green pus was removed. Further exposure revealed a definite capsule, in which was placed a soft rubber drain and 500,000 units of penicillin were instilled. Improvement was noted the following day and another 500,000 units of penicillin were instilled into the cavity, but one hour later the patient had a convulsion and the temperature increased to 103.2.° The drain was removed and the patient's condition improved rapidly. Culture of the material from the abscess revealed B. coli communis and Streptococcus viridans.

Fourteen days after the neurosurgical operation a right endaural radical mastoidectomy was performed. Audiometric examination the day before surgery revealed a 60 decibel loss on the right with normal hearing on the left. The appearance of the ear was unchanged, although the patient had received intramuscular crysticillin during the entire hospital stay, varying from 300,000 to 600,000 units twice daily. A fistula test was positive.

Upon entering the antrum with a perforating burr thick pus under pressure was encountered. The mastoid and middle ear contained a large cholesteatoma extending into the mastoid tip, and granulations, which were entirely removed. The facial nerve and its Fallopian canal were intact. The lateral sinus plate and tegmen showed no signs of breakdown. A small fistula was noted in the horizontal semicircular canal. The entire cavity was grafted with split thickness skin grafts from the thigh. Culture of pus from the mastoid showed B. coli communis, one of the organisms found in the brain abscess. Streptococcus viridans was not cultured, possibly because of continuous penicillin therapy.

Postoperative recovery was uneventful; the patient was discharged from the hospital on the sixth postoperative day after mastoidectomy (the twenty-first hospital day). When last seen on November 1, 1952, he was normal in every respect and had been back at work for several months. He has no memory, however, of events immediately preceding and following the first operation. The ear has been dry since two months following surgery.

DISCUSSION

DR. THOMAS C. GALLOWAY: I think this abscess should have been cared for just as well through the radical mastoid approach, thereby subjecting the patient to only one operation. I wonder how Dr. Theobald feels about that.

DR. SHERMAN SHAPIRO: How long was the drain kept in the abscess? Does Dr. Theobald feel that the cavity is sterile and that the situation will not recur?

DR. PIERCE W. THEOBALD (closing): I think Dr. Galloway is correct and that the abscess could have been taken care of just as well through a radical mastoid wound as via the neurosurgical route. On the other hand, we younger men do not see the cases that the older men in the Society have seen, and I feel, therefore, that the neurosurgeon is better trained to perform intracranial procedures. During my training I saw only one case of supposed otogenic brain abscess that was operated upon through the mastoid, at the insistence of the neurosurgical service, and it turned out that the patient did not have a brain abscess.

The drain in this case was left in the abscess cavity for only two days and, with the occurrence of acute increase in fever and convulsions, was removed. As to whether the abscess cavity is sterile and completely obliterated I cannot say, nor do I think anyone else can. The patient has been well since March, 1952, nearly nine months, but whether he will remain so I cannot say.

Late Development of Laryngeal and Pharyngeal Carcinoma In Previously Irradiated Areas

PAUL H. HOLINGER, M.D.

AND

WILLIAM F. RABBETT, M.D., LT. (M.C.) U.S.N.R.

Three cases of malignancy of the pharynx and larynx are reported in which the tumor developed in an area previously irradiated for a benign process. A review of the literature reveals reports of nine similar cases; three are males and eight are females in the total of 12 cases. All had received irradiation 16 to 32 years before development of the neoplasm; seven received irradiation for tuberculous cervical adenitis and five for thyrotoxicosis. Cases of carcinoma of the skin developing in a previously irradiated area are well known. Recently, malignancies have been reported developing in deeper structures previously treated for benign lesions. This has occurred in bones, as in the development of bone sarcomas in areas treated three to 12 years previously for tuberculous arthritis, and carcinoma of the cervix developing ten or more years after irradiation treatment for a benign lesion of the cervix.

While statistically the series of 12 pharyngeal and laryngeal malignancies is small, it would be difficult to believe that the association of the irradiation and the development of the tumor is merely coincidental. Since irradiation therapy was more common for benign conditions of the neck several decades ago, more of these cases may be seen in the near future.

DISCUSSION

DR. M. F. SNITMAN: This spring at the National Cancer Society meeting, Dr. Lauren Ackerman called attention to the importance of continued observation of patients who have received irradiation therapy of oral carcinoma, because in their experience it is not unusual to find at a later date the development of a new lesion which had no direct connection with the original primary one. One might ask whether the second primary lesion was due to the irradiation effects or to the same condition that caused the first. In our observation of patients treated for pharyngeal carcinoma who have a five-year cure, we have as yet not found a new development of carcinoma in the pharynx, although we must admit that a period of five or ten years is insufficient. We are well aware of the cancercidal effects of irradiation, and I think Dr. Holinger's paper should bring to mind the possible effects of irradiation therapy on the overgrowth of the pharyngeal tonsil causing Eustachian tube obstruction.

DR. PAUL H. HOLINGER (closing): Dr. Snitman's remarks about irradiation of tissues in the postnasal space are important. When one sees such irradiation administered indiscriminately, sometimes repeated several times without knowledge of the previous dosage the patient may have had, one recognizes a similarity in the history between these patients and the ones herein presented. When one finds an atrophic rhinitis and pharyngitis sicca with extensive scarring and crusting, and obtains the history of several "courses" of postnasal irradiation, one must be concerned about possible late development of carcinoma in such cases.

Sudden Death Following Radical Mastoidectomy

GEORGE E. SHAMBAUGU, JR., M.D.

A 60 year old white male was referred to the Ear, Nose and Throat Clinic of the Montgomery Ward Clinics on June 3, 1952, because of constant drainage from the right ear for about 55 years, with pain in the right mastoid for many years. Examination re-

vealed an anterior attic perforation in the right ear with a foul purulent discharge. Attic irrigation on three successive visits obtained cholesteatomatous material. Because the discharge and pain persisted, surgery was advised.

This man had been seen previously in the diagnostic, circulatory and gastrointestinal clinics. On the basis of history and electrocardiogram, a diagnosis of old coronary occlusion was made. There were symptoms suggesting peptic ulcer. X-ray studies showed a hiatus hernia, no evidence of gastric or duodenal ulcer. Esophagoscopy was attempted in the medical G.I. clinic on August 6, but the patient vomited 120-150 cc of coffee ground material and the examination was discontinued. Ten days later esophagoscopy revealed an esophagitis of the lower end of the esophagus. Three days later he was given medical clearance for mastoid surgery.

On September 9, a Bondy modified radical mastoidectomy was performed by the endaural route. The operation was begun under local anesthesia with three grains of nembutal and 1/6 grain of morphine for sedation. Due to discomfort, the operation was continued under pentothal anesthesia, a total of one grain being given in two and one-half hours. A cholesteatomatous sac was found to occupy the attic region.

This man's blood pressure which had varied between 130 and 140 cystolic and 80 and 90 diastolic in the previous three months, was 110/60 at the beginning of anesthesia and 90/50 at the end of the operation, but with a good pulse of 80 and respirations of 20. He returned to bed at 5:00 P. M. in good condition; his blood pressure remained about the same until at 6:30 it fell to 78/50, respirations had increased to 24, and the pulse to 126. At 7:00 he responded to questions and continued to respond until 8:00 P. M., when the blood pressure fell to 80/60, he became unconscious and ashen gray, with a pulse of 120. Oxygen and glucose saline were given intravenously. At 8:30 he was given a vasopressor; at 8:45 he suddenly vomited about a quart of coffee-ground material and five minutes later suddenly died.

Unfortunately, autopsy permission was refused by the only relative, a brother who did not come in until next morning, when he arrived inebriated and abusive. Therefore, we can only speculate that this man presented the picture of fatal circulatory collapse three hours and fifty minutes after return to bed from surgery, preceded by a low blood pressure that began to fall during surgery. The copious coffee-ground vomitus, with the long history of gastric symptoms suggests a bleeding lesion of the stomach, either a varix,

an ulcer or a neoplasm. Certainly the blood lost during the mastoidectomy was hardly sufficient to be a factor in the circulatory collapse.

It is worth mentioning that this is the first surgical death in approximately 3,500 endaural procedures at Wesley Hospital during the past eleven years. It seems unlikely that the surgical procedure was the immediate or contributing cause of death in this case.

DISCUSSION

DR. MARY KARF: The history of this patient is of interest. There had been several coronary episodes, the first in 1937 when he was bedridden for six weeks, and another in 1948, following which he was unable to work for one year because of dyspnea, chest pain and orthopnea. The electrocardiogram on his first admission to the clinic showed no diagnostic deformities; however, this was not repeated. Several further visits confirmed the diagnosis of esophageal hiatis hernia, chronic pyelonephritis, chronic otitis media with cholesteotoma. On August 18 the medical department gave clearance for surgery. Dr. Shambaugh has given the details of the subsequent course.

Although autopsy was not performed, one can postulate that a coronary thrombosis or circulatory failure occurred. When, in relation to the surgery, this did occur and whether it might have been avoided is a question. Could it have occurred when the local anesthetic with adrenalin added was absorbed? The first blood pressure reading showed a fall of 50 points of systolic pressure. This might have been due to the secondary reaction of adrenalin, to the heavy preoperative sedation, to reaction to local anesthesia, or to coronary occlusion. A blood pressure drop in a hypertensive patient may be significant for he may need a working head of pressure for adequate circulation, especially if the coronary circulation is already impaired. This patient's hemoglobin was 11.1 grams or 71 per cent. This anemia may have placed a further load on the oxygen carrying power of the circulation and on the small cardiac reserve. Certainly the depression of circulation caused by pentothal anesthesia of 170 minutes duration added its weight. Although no artificial airway was used the anesthetist stated that the breathing was unobstructed throughout. Occult hypoxia may have existed to add another factor.

The case brings up several points: First, the need for more thorough preliminary evaluation of the cardiac status. Perhaps had the entire team been cognizant of this man's poor cardiac reserve, surgery might have been delayed or premedication altered, or attempts

made to avoid hypotension during and after surgery. One might postulate that the use of nor-epinephrine during the surgical procedure and later to increase peripheral response of coronary circulation might have changed the result. We have seen several cases of massive proved coronary infarction which have responded when the shock factor occurred, by treatment with controlled nor-epinephrine intravenously. Another point is the need for a planned general anesthesia with control of the airway. This is especially indicated when general anesthesia is used for procedures on the head and neck.

Dr. George H. Woodruff: Dr. Shambaugh mentioned a possible gastric hemorrhage. It could be that the restlessness under anesthesia was due to such an occurrence. Many patients who hemorrhage are restless and this might have been a factor in the unsatisfactory local anesthesia.

Dr. George E. Shambaugh (closing): I wish we could call this something else than death following radical mastoidectomy, but unfortunately that is what it was. We do about 50 per cent of our radical mastoidectomy operations under local anesthesia. When necessary we supplement with pentothal and a very small amount of pentothal will carry the patient along quite satisfactorily after the premedication used for the local anesthesia. We have not felt the need of intubation of these cases. The radical mastoid operation itself as performed now is certainly a procedure with a minimum of shock and a minimum of risk. This patient died evidently of some associated condition which was not thoroughly understood in advance. Our fault lay, I think, in not being careful enough in the preoperative workup. The patient had been given medical clearance for surgery, but nevertheless apparently died of a cardiac or gastric condition that was present before surgery.

Radical Neck Dissection for Hodgkin's Disease

Irwin D. Horowitz, M.D.

There is a growing tendency to regard Hodgkin's disease as not a generalized disease of the lymphatic system, but rather as unicentric in origin. This theory carries the implication that the disease may be curable. Surgery is becoming an increasingly used method, either as an adjunct to irradiation or as the primary therapy. Reports of median survival rates of five years, or cases living six to 14 years disease-free after surgery, tend to show that this type of ther-

apy, is definitely helpful. Even if not curative, excision of the primary lesion seems to favorably alter the subsequent course of the disease.

Indications for surgical intervention are strictly limited: (1) The lesion must be isolated to one portion of the body; (2) Its surgical removal must be technically feasible; (3) there must be no systemic manifestations of the disease such as fever or anemia. These criteria were met in the case of the patient to be described.

This 34 year old white female, in apparent good health, consulted her physician because of a painless swelling of the left neck. He thought it might be a non-specific adenitis, as the patient was recovering from a cold. One week later the mass was undiminished in size and its removal was advised.

Physical examination revealed a mass about in the left posterior triangle of the neck just above the clavicle. It was about 4 centimeters in extent, non-tender, slightly movable, and not attached to the overlying skin. There was no evidence of other adenopathy. X-rays of the chest and abdomen were negative, as were blood tests.

On April 11, 1952, the tumor mass was excised. This was lying on the floor of the posterior triangle; the clavicular head of the sternocleidomastoic muscle had to be transected to mobilize the mass. The pathology report was Hodgkin's disease.

On April 18, 1952, a radical neck dissection was performed. Careful histologic study of the specimen did not reveal any other foci of disease.

The postoperative course has remained uneventful, and the patient is disease-free when checked by palpation, x-ray, maintenance of weight, and lack of anemia.

DISCUSSION

DR. STANTON A. FRIEDBERG: The hopeless outlook in Hodgkin's disease has recently inspired more drastic surgical attempts to remove unicentric foci of the disease. However, in a series of 250 patients recently reported from Memorial Hospital, only two had lesions which definitely could be regarded as representing isolated foci. It must be remembered that no one can foresee whether the disease will remain limited or whether radical treatment will result in long-term survival.

It is true that Hodgkin's disease usually runs a rapid course but it is also true that numerous individuals survive for many years as a

result of effective irradiation to new areas of involvement as they appear. In a series of lymphomas currently being studied, our experience has been that isolated foci in the upper passages have been cured, only to be followed by reappearance of the condition in the thorax or abdomen after varying periods of latency.

Any attempts to reduce the mortality in Hodgkin's disease are deserving but must be judged from the point of view that this is a systemic disease and is usually of multicentric origin. Because of the radiosensitivity of the lymph nodes in this and other lymphomas, the use of irradiation after surgery is more logical and effective than in almost any other form of malignancy. Since all surgically-treated patients are also treated by irradiation, the results will be difficult to evaluate, particularly since long-term survivals with irradiation alone are not uncommon.

DR. M. F. SNITMAN: I do not wish to detract from the value of radical surgery in localized types of lymphoblastoma, but the question in this case is the possibility of error of diagnosis. No two pathologists will always agree, especially when dealing with tumors of the lymphatic system. Dr. Horwitz will remember the patient we observed about two years ago with the large node in the right supraclavicular triangle, which was biopsied, and the pathology, department made a diagnosis of an atypical type of lymphatic pathology, but we felt it was a definite lymphoblastoma. The patient was sent for irradiation, and the pathology department then said it was not a lymphoblastoma but simply a form of reticulum cell hyperplasia; consequently irradiation was stopped and the patient is well to date.

DR. IRWIN D. HORWITZ (closing): There is a growing tendency to group all the lymphomas together, whether reticulum cell sarcoma, lymphosarcoma, large cell or small cell; to put all the lymphomas together except the leukemias. These have a different course and they do seem to be unicentric. The reason they are thought to be multicentric, perhaps, is that by the time the doctor sees the patient the disease is disseminated. This case was unusual, in that it came to surgery in seven weeks. Most cases go a year or more before they are seen or diagnosed. The disease is painless in most cases, and "watchful waiting" is carried on too long.

There are only three types that can be operated on; the neck, the groin and the mediastinum. Operation on the groin consists of radical dissection along the lumbar aorta. The neck we know about. The mediastinum has been reported in two series that I have found;

one was a series of five, the other an Army group. These were all fatal within a short time.

The point is that whether surgery or x-ray is used, palliation should not be attempted. Either radical surgery or what the roentgenologists like to call "obliterative x-ray therapy" should be used. The statistics, such as they are, show more cures at the median survival rate which, to me, is more important than the average survival rate. The median survival rate in one group of lymphosarcomas was raised from 1.2 years to more than 5 years. Half the patients lived more than five years, whereas formerly half lived only 1.2 years. That is statistically significant.

ACTH in Lethal Granuloma of the Nose and Face: Report of a Case

E. W. Hagens, M.D.

Normal Parry, M.D.

AND

David Markson, M.D.

The case presented was a man aged 60, who complained of left-sided nasal obstruction for five months. There was also a subcutaneous nodular swelling at the root of the nose on this side, extending toward the eye and cheek. Examination considerable ulcerative tissue involving the left inferior turbinate. Pressure here produced movement of the subcutaneous lesion at the root of the nose. Biopsy of the intranasal tissue was reported as "necrotizing inflammation of fibrous connective tissue."

External operation was performed and part of the lesion was removed, the remainder being found to infiltrate the subcutaneous tissues. Again the pathologist reported inflammation. The wound healed but broke down after six or eight weeks, forming a large necrotic ulcer with rolled edges. The patient was again hospitalized, and on the basis of the case being a lethal granuloma, ACTH was administered. Treatment was stopped after twenty-five days as the lesion was already healing. Complete recovery occurred shortly afterward, and is still present seven months after cessation of therapy.

DISCUSSION

DR. ALFRED LEWY: Again our Society has scored a notable first; I believe this to be the first case of lethal granuloma cured by any treatment whatsoever. I can record a similar case, also seen by Dr. Hagens when he was my associate at County Hospital. This was an 11 year old girl who, following scarlet fever in March, had an antral window done in April. When I saw her in July there was swelling of the face that obliterated the nasolabial fold on the left side, and a purulent discharge from the antrum. After a few irrigations I performed a Caldwell-Luc operation. The process continued to progress in spite of all forms of oxidizing agents. Dr. Jaffe and Dr. Fisher, an expert mycologist, thought it was a microaerophilic streptococcus infection. Inoculations were done on guinea pigs and rabbits; the animals survived but developed nodules. The nodules yielded Streptococcus hemolyticus in one case, and in the pus were found rods and Spirilla; in one case a blastomyces was recovered. We thought we did not have conclusive evidence of any particular infection. The pathologic specimens were submitted to several men, including Dr. Davidsohn at Mt. Sinai Hospital, and all reports came back simply granuloma.

One interesting thing in this case was this 11 year old girl, at the time of death, measured six feet in length—much larger than either of her parents, and at the time I attributed that to pituitary irritation. The lesion had extended into the sphenoid and beyond. She died of uncontrolled hemorrhage. I could not see that anything we did had any effect on the disease.

I reported this case to the Triological Society in 1937, calling it a gangrenous osteomyelitis. I think the name lethal granuloma is more suitable, and covers practically all the cases reported.

DR. GEORGE H. WOODRUFF: I wish Dr. Hagens would say a few words about the rationale of the use of ACTH in this case.

DR. THOMAS C. GALLOWAY: We know so little about so-called lethal granuloma that we are perhaps not always able to diagnose it. Sometimes confused are microaerophilic or anerobic streptococcic infections or osteomyelitis which progress in spite of all that is done.

A patient came to us last year with a granulating lesion involving the right alveolar region and maxilla, with anerobic sinus infection and osteomyelitis. There was infiltration of the conjunctiva and sclera on the right side. Skin lesions had previously appeared and a tentative diagnosis of lupus erythematosus disseminata had been made. We took biopsies from the antrum which we opened widely

by removing the floor by the use of surgical diathermy, and the lesions were diagnosed by our own pathologist as nonspecific granuloma. (Later I found there had been disagreement in the pathologic laboratory.) This patient was put on ACTH and cortisone up to 200 mgm per day, and the lesions healed completely except for one area of about 5 mm. The dose was cut down and the lesions began to recur, granulations increased and there was destruction of the palate. Another biopsy was reported as nonspecific granuloma and the patient thereafter had little result from ACTH. Dr. Williams saw him and biopsy was diagnosed as probably a lympho-epithelioma. Our pathologists now agree that it is a malignancy which may be a reticulum cell sarcoma, a lympho-epithelioma, or an undifferentiated carcinoma. The case is not doing well even with irradiation. I ligated the external carotid yesterday.

The interesting point is that this lesion at one time did almost heal with ACTH. Has there been any such experience with malignancy, or is it possible that this is a type of lesion which did secondarily undergo malignant change? It did not metastasize early but now there are glands rather extensively involved. This is a puzzling disease, and Dr. Hagens is to be congratulated on his results.

Dr. Elmer W. Hagens (closing): In answer to Dr. Woodruff, many concepts have been advanced in the tremendous literature, the sum and substance being that this condition is to be correlated with allergy, in its widest sense. If it is an allergic condition, its response to ACTH and cortisone may be logical.

A. G. Rawlins of San Francisco, in the Annals of Allergy for July-August, 1952, says that edema is the main pathology in the allergic state and that clinically allergic edema is located in the jell of connective tissue. The enzyme hyaluronidase liquifies the neutral mucopolysaccharide jell of connective tissue and thus produces edema. Hyaluronidase is present in or around connective tissue at all times. This enzyme is liberated from the damaged tissue in large amounts in allergic inflammation of the skin. Thus in allergic inflammation hyaluronidase is available for the production of allergic edema. In vivo, ACTH or cortisone neutralizes hyaluronidase and restricts allergic edema. Normally there is an equilibrium in the enzyme action which builds up and breaks down hyaluronic acid. Thus, even while under stress a firm normal connective tissue jell is maintained in nonallergic individuals. In an allergic state there is either breaking down of enzyme, over-balancing the building-up enzymes, thus requiring an excess amount of cortisone to maintain equilibrium, or possibly there is dysfunction in the autonomic nervous system and

hormonal system in which not enough cortisone is produced to maintain equilibrium.

He agrees with Williams that many allergic humans do form sensitizing antibodies to small amounts of so-called harmless substances. In many the antigen-antibody union does produce stress with tissue damage and edema; in others the same tissue damage and edema will develop from other physical or emotional stress and without the antigen-antibody mechanism. That mechanism is only one specific type stress that produces pathology in allergic humans. Therefore, it is the fact of its being allergy in its broadest sense that gives this treatment its chance. Rawlins has this hypothesis; others say they do not know. All I can say is that this patient has been well for seven months. That may be a relatively short time, but it is remarkable that he has been so well during this time.

MAGAZINE SHOW 1952

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December, 1952

JOHN J. SHEA
1889-1952

JOHN J. SHEA, M.D.

1889-1952

John J. Shea died in New Orleans, Louisiana on November 28th, 1952 following an operation for malignancy of the pancreas. He was actively engaged in his practice until but a few weeks before his death, having also participated in the examination of the American Board of Otolaryngology, of which he was Vice-President, early in October, a typical example of his unselfish devotion to his duty as a physician and an educator dedicated to the advancement of the science of otolaryngology.

Dr. Shea was born in Memphis, Tennessee on January 17, 1889, where he received his early education, later attending high school and college at the Christian Brothers College from which he received his Bachelor of Arts Degree in 1906. He received his M.D. degree from New York University and Bellevue Medical College in 1911.

He served his internship at St. John's Seaside Hospital, Staten Island, N. Y., Manhattan Maternity Hospital, New York, and Bellevue Hospital, New York from 1911 to 1913. His post-graduate training was obtained at the New York Post-Graduate School and Hospital in 1914 and 1915, and the University of Paris, in 1918 and 1919.

He was commissioned as a First Lieutenant in the United States Army Medical Corp in 1917 in which he saw service at Camp Meade, Md., Red Cross Hospital No. 7 Juilly Seine et Marne and U.S.A. Base Hospital No. 57, A.E.F. Paris, France. He was honorably discharged in 1919 with the rank of Major. He later held the position of examiner for the Department of Aeronautics from 1925 to 1932.

After his discharge from the military service he returned to Memphis where he engaged in the practice of otorhinolaryngology and maxillo-facial surgery in the offices of Drs. Ellett and Farrington. In 1926 he established the Dr. John J. Shea Clinic, where he practiced until the time of his last illness.

He was a prominent member of the Staffs of the Baptist Memorial Hospital, St. Joseph Hospital and the Memphis Eye, Ear, Nose and Throat Hospital. He was also a consultant for the Campbell Clinic and the Kennedy Hospital.

His intense interest in otolaryngology and his indomitable energy were manifested by his regular participation in the activities of the

numerous national medical societies of which he was not only a member but by which he had been honored by election to many high offices. He was a Fellow of the American Medical Association (Secretary, Section of Otolaryngology 1931-32-33; Chairman 1934-35) Southern Medical Association (Secretary of Section O. & O. 1920-21-22; Vice-chairman 1923 and Chairman 1924). American College of Surgeons 1922. American Academy of Ophthalmology and Otolaryngology (Vice-President 1924; Member of Council 1929-30-31-32). (President-Elect 1953); American Laryngological, Rhinological and Otological Society (Council 1937-38-39; President 1948-49); American Otological Society (Council 1943-44-45); American Laryngological Association (1st. Vice-President 1944-45 and 47).

He was a prolific writer, having published ninety-four papers on all phases of otorhinolaryngology, maxillofacial surgery, allergy, and several on diseases of the hematopoetic system. He was likewise credited with devising the "Shea Tube' for use in the postoperative treatment of nasoantral surgery in children.

On July 5th, 1917 he married Catherine Flanagan, his constant and delightful companion at the meetings of the American Board of Otolaryngology and of the national otolaryngological societies, with an attendance record second only to his own. There are six children, Jeanne (Mrs. Wm. A. Leatherman), Catherine (Mrs. W. L. Roberts), Ellen (Mrs. L. K. Thompson, Jr.), Mary (Mrs. David F. Dixon), Martin and John, Jr., the last having selected the practice of medicine for his career and the specialty of otolaryngology also, in emulation of his father. They enriched his life by giving him fifteen grandchildren.

If he could be said to have had a hobby it was above all his family, of which he was inordinately proud, and which brought him the greatest happiness. He was never more radiant than when he was showing the many photographs of his wife, his children and his grandchildren, and relating interesting anecdotes of their everyday lives, and the great family dinners which were a part of the Sunday ritual at his home.

He was an ardent Catholic whose religion meant everything that it should mean to a man and to a physician. He was a man of the highest principles, of great charity to his fellow men, a man of tolerance and understanding of the shortcomings of others, as well as his own ineptitudes. Those of us who had the privilege of being associated with him on the American Board of Otolaryngology appreciated his honesty, his sagacity, his straightforwardness and his

unswerving sense of duty towards his fellow members and the candidates alike. His disposition was always cordial and ebullient, his sense of humor unfailing at all times. He was the exemplary 'southern gentleman'. It was most regrettable that he did not attain the honor of serving as President of the Board, being President-elect at the time of his death, as he was also President-elect of the American Academy of Ophthalmology and Otolaryngology.

The greatest tribute to his memory was the last which was paid to him by the hundreds of grieving patients and friends of all walks of life and the large number of fellow physicians who were in attendance at his Funeral Mass in Memphis.

We shall miss John Shea, but he shall always be remembered as the epitome of kindliness and loyalty and as a man of great professional achievements and highest integrity.

B. J. McM.

Abstracts of Current Articles

EAR

On the Genesis of Otosclerosis.

Nylen, C. O., and Hylen, Bengt.: Journal of Laryngology and Otology 56:55-64 (Feb.) 1952.

The authors comment on the fact that there have been relatively few papers on the genesis of otosclerosis in the literature of recent years, partly because of the great difficulties of such investigations, but especially through concentration on the surgical treatment of this disease, which has taken the main interest for the moment. Ankylosis of the stapes was mentioned by Valsalva in 1735 and Toynbee was the first to use the term "otosclerosis" a hundred years ago. The pioneers in the histopathology of this disease up to 1920 have described most of the principal features, but those important researches have not given us the fundamental cause of otosclerosis. The etiological investigations must be continued with energy and enthusiasm.

The authors comment on some of the new statistical and histopathological points, which are important to our augmented knowledge about the genesis. They refer to Guild's statistics that about 8 per cent of the white race suffer from otosclerosis, and that the disease is relatively uncommon among young people. The authors, however, are inclined to agree with Fowler, Jr's. figures that a fair estimate as to the white race would be nearer 10 per cent for males and 20 per cent for females, if undetected or "potential" otosclerosis is included, except that they think that the distribution between the sexes may be more uniform.

They emphasize the fact that the blood circulation in the normal bony capsule of the labyrinth is still unknown. Their research has aimed at and is continuing to try and clarify this. They believe that the hormonal possibility as a cause of otosclerosis should be intensively studied. They suggest that two factors each separately, or perhaps more probably in combination, are of decided importance for the genesis in developing otosclerosis:

1. A particular vascularization of the otic capsule.

2. A metabolic disorder, mainly owing to imbalance of the internal secretion glands.

At present the most plausible disturbance seems to be a slight, primary or secondary, dysfunction of the parathyroid glands, influencing the metabolism especially of enzymes and mineral salts.

BOIES.

Otosclerosis: A Closed Method of Fenestration.

Garson, Philip: *Journal of Laryngology and Otology* 56:65-70 (Feb.) 1952.

The author reports his experience with 35 cases of fenestration for otosclerosis in which he uses a graft of conjunctiva to cover the fenestra and thus avoids a plastic flap which alters the anatomy of the middle ear and creates an open cavity.

He also believes that he improves his technique through the use of pentamethonium by the anesthetist to lower the blood pressure and to prevent bleeding. He believes this is the most important single factor contributing to the improvement in his technique.

In some of the cases the incus was not removed but the author now believes that the operation can be shortened by removing the incus and that the result is just as satisfactory as it was when it was left in position.

The essential advantages of this closed method is the fact that the convalescence is short and the risk of a chronically discharging ear is almost eliminated.

BOIES.

Nuclear Jaundice and Deafness.

Gerrard, John (Birmingham): *Journal of Laryngology and Otology* 56:39-45 (Jan.) 1952.

A study was made to determine the possible relationship of perceptive deafness to severe neonatal jaundice and to Rh incompatibility or to prematurity. Residents of a School for the Deaf were examined.

It was found that deafness was associated in 5 and possibly 6 of 407 children with kernicterus due to Rh iso-immunization.

In a further study of 17 cases there was a comparable perceptive deafness associated not with Rh iso-immunization but with a high incidence of severe neonatal jaundice and prematurity.

BOIES.

The Organ of Corti by Phase Contrast Microscopy.

Katsuki, Y., and Covell, W. P.: The Laryngoscope 63:1-17 (Jan.) 1953.

The authors studied freshly isolated pieces of organ of Corti from normal guinea pigs by means of the phase contrast microscope and present information and excellent photomicrographs of its finer cell structure.

They note that the hair cells are the most fragile components of the organ of Corti and disintegrate rapidly. The cells appeared quite clear with granules, mitonchondria and a ground substance of a non-refractile type. It is possible that fibrils were present but were undetected.

The tectorial and basilar membranes were found to be composed of fibrils with intervening interfibrillar substance that is probably of different chemical composition in the two structures.

Several features are presented which throw some doubt on the value of Hensen's cells as supporting elements for the organ of Corti. It was shown that they had no firm anchorage to the reticular membrane or basilar membrane.

The course of the peripheral nerve fibers in the organ of Corti was studied. It was found that the nerve endings of the external hair cells are of one type while two types were observed in the internal hair cells.

The recent findings of Hilding and DeVries regarding the tectorial membrane were confirmed.

SENTURIA.

Glomus Jugularis Tumor of the Middle Ear. A Case Report.

Land, Richard N.: Radiology 59:70-78 (July) 1952.

A case of glomus jugularis tumor of the middle ear is reported. Symptoms had been present for three and a half years. The roentgen findings of extensive bone destruction in the mastoid process and petrous portion of the temporal bone as the result of pressure necrosis as demonstrated in six radiographs with descriptions. The differential diagnosis from benign processes is discussed. Two photomicrographs illustrate the type of tumors. A brief case report including outline of treatment used is given. A brief review of the discovery and previous reports of this unusual tumor is made. Fifteen references are cited.

JORSTAD.

Functional Studies on Labyrinthine Fenestration in Animals—II.

Benedict, Margaret R.: Laryngoscope 62:1293-1315 (Dec.) 1952.

The monaural hearing threshold of a series of monkeys was determined by conditioned response technique both before and after the ossicular chain was interrupted by removal of the incus. The hearing losses caused by this operation averaged 44.6 db for the frequencies of 150, 500, 1000 and 4000 cycles per second. A fenestration operation was then performed upon the ear from which the incus had been removed and the monaural thresholds were obtained periodically and compared with pre-fenestration limens. At the time of death nine to fifteen months after fenestration over half of the animals had maintained hearing improvements averaging between eight and ten db at each of the four frequencies tested, although variability of the hearing changes was very great. Microscopic examination of the sections of the fenestrated ears showed no clearcut correspondence between the ear structure at the operative site and any pattern of functional hearing changes.

This investigation indicates that the post-operative hearing improvement in this series of animals was independent of the open or closed fenestra. The author suggests the possibility with its many implications, that the improvement of hearing resulting from the fenestration operation, may be related to anatomical changes produced by the operation in and around the middle ear.

SENTURIA.

NOSE

Respiratory Air Currents (The Semon Lecture).

Proetz, A. W.: Jour. Laryng. & Otol. 67:1 (Jan.) 1953.

Abstract Reprinted from The Lancet, No. 22, 1952, *p.* 1030, *with permission.*

"The more violent air currents emerge as the coughs and sneezes that are of such popular epidemiological significance; but the gentler currents in the upper repiratory tract can also be important. Dr. Proetz described some of the physical properties of air currents in general. If smoke from a cigarette is blown through a straight glass tube of uniform bore, no particles are deposited on the tube; but if the tube is bent or constricted, there is a turbulence in the smoke and a deposit is rapidly formed just beyond the bend or constriction. A homely example of this may be found in the smoker who has an obstruction in one side of his nose. If he blows the smoke out through his nose there is a deposit of particles beyond the constriction, and

the vibrissæ (or long hairs) just inside the nostrils become stained brown.

"If the nose is healthy, the air currents pass into the nasopharynx without obstruction. The nasal airway in man is normally only about 1 mm in width, and the nose gets its capacity through height rather than width. The air currents in the nose are affected by three things: the size and direction of the external nares; the general shape of the nose; and the difference in size between the anterior and posterior nares, the latter being larger than the former. Normally the nares are directed downwards so that, on inspiration, the air first of all passes straight upwards; it then passes through the upper part of the nose in a gentle curve; and it leaves the nose through the posterior nares to enter the nasopharynx. This inspired air does not come into contact with the ostia of the sinuses in the middle or the superior meatus. As the air passes downwards from the nasopharynx to the larynx, there are turbulent areas at the back of the mouth, under the palate, and round the epiglottis; and it is interesting to note that these are the very situations where aggregations of lymphoid tissue are found. During expiration there are in the normal nose eddies of air under the middle turbinates, and the ostia of the sinuses are, therefore, reached by this air alone, after it has been moistened. There is very little air exchange in the sinuses themselves.

"Abnormal air currents may be caused by various lesions—by deviations of the nasal septum, by polypi, by swelling of the turbinates, by enlarged plicæ septi, or by adenoids. The air currents in the nose normally pass through its upper, and not its lower, part, and a swelling high in the nose, such as may occur in a common cold, will leave only a small passage for the air through the lower part of the nose. Air then passes straight back and impinges against the nasopharynx, where the adenoids are situated. This produces the dry spot at the back of the throat in the early stages of a cold. A deviation of the septum may often have to be corrected by surgery, but not always. Sometimes the turbinates will enlarge on the unobstructed side, and so adapt themselves to the abnormal condition of the septum. If a submucous resection is performed in such a case, there will be too much room on the unobstructed side when the septum comes to lie in the midline; and there will be a constriction on the other side where the straightened septum lies against the enlarged turbinate. In such a case, a dry area will develop, with subsequent stasis and liability to infection. The remedy is, therefore, not always surgical. When there is only a low spur on the septum, this is streamlined and air can pass above and below it. There is therefore no interference with the air currents and no harm is done. However,

should the patient with a low spur catch a cold there will also be swelling in the upper part of the nose with considerable obstruction. Polypi in the superior meatus of the nose will interfere with the air currents, but polypi in the middle meatus, although they will have to be removed for other reasons, have an almost negligible effect on the air currents. If the middle turbinate is removed there is practically no change in the air stream; but if both middle and inferior turbinates are removed, what is tantamount to atrophic rhinitis develops, and true atrophic rhinitis soon follows. Although there is then a much wider air space than normal, the patient complains of nasal obstruction, because there is eddying of air in the nose on inspiration, with deflection of the normal currents.

"Dr. Proetz illustrated his remarks with two excellent films which demonstrated the behaviour of smoke in various kinds of tubes; the mucous blanket being moved by the cilia; red cells being tumbled along at a great pace; the stasis that follows drying; and the normal air currents and their variation under different conditions."

The Histo-Pathology of Sinusitis.

Lucas, H. A.: Journal of Laryngology and Otology 56:480-489 (Oct.) 1952.

This study differs from the usual recital of well established facts concerning histo-pathologic changes in the mucosa of various sinus conditions. Emphasis is given to the role of the vascular system in the nose. This is composed of arteries emerging from the bony wall which terminate in capillary networks in the sub-epithelial layers of the mucous membrane. These capillaries connect with veins which in the deeper parts of the mucosa form plexuses, the so-called erectile tissue, which differs from true erectile tissue in that there is no arterio-venous anastomosis. The walls of these venous plexuses contain smooth muscle arranged in a spiral manner around them and the system is closed by sphincter muscles which lie deep, close down to the bone.

This muscular vascular system is controlled by the autonomic system. This is shown by the action of drugs introduced into the nose. If stimulation in the form of heat is applied to the superior cervical ganglion, a severely occluded nose can be rapidly cleared so that there is a free airway. It is difficult to see how this sudden clearance could occur except by the relaxation of these sphincter muscles.

This system is also acted upon by hormones. The best clinical example is the rhinitis and sinusitis which occur at the menopause, and the changes observed in the nose during pregnancy.

This vascular system is the means whereby the normal nose reacts to all environmental changes. The absence of this function is well seen in atrophic conditions. The congestion of the erectile tissue is present in all cases of "stuffy nose."

The paper contains an excellent discussion of non-suppurative sinusitis and a further consideration of how the autonomic nervous system is fundamentally the means whereby the organism reacts to its environment, insofar as the nasal space is concerned.

He concludes that the etiology of sinusitis is complex. The demonstrable changes arise from the reactivity of the reticulo-endothelial system. This is invoked by an antigen-antibody response to infection and inhaled protein material which is antigenic. The author regards allergy as a dysfunction of this type of reaction and from the nature of things it is frequent. The cellular reaction seen in these cases is the result of this antigenic stimulation and these reactions are characteristic, though mixed types occur. Coincident with this reaction is an autonomic imbalance of the erectile tissue. Immunologists regard this as the result of the antigen-antibody response; some others regard it as an inherited condition which precedes, and predisposes towards this type of disease in the nose and sinuses.

BOIES.

Endocrines and the Nose.

E. Watson-Williams: *Journal of Laryngology and Otology* 56:29-38 (Jan.) 1952.

The author apparently believes that the majority of cases of atrophic rhinitis are related to a diminished thyroid activity. In 1934 he had analyzed a group of 1,382 cases of atrophic rhinitis as to their probable etiology. In a group which were thought to be due to infection there were a total of 352, and in a group in which he thought there was a definite relationship to a "goitre" condition there were 764, plus an additional 266 which seemed to be clinically similar. In the group due to infection, the author believes that syphilis is the cause in the majority of cases. The treatment in the "goitre" cases consisted of administering small doses of thyroid substance over a long period of time. Good results are reported. In

the cases with hyperthyroidism a hypertrophic rhinitis characterized by engorged turbinals, excess of water secretion and the tendency to sneezing attacks was noted.

The second half of this paper is concerned with the relationship of the reproductive system and the nose. Menopausal rhinitis is discussed. Case reports are offered on the relationship between sexual activity and nasal symptoms.

BOIES.

Osteomyelitis of the Maxilla.

Macbeth, Ronald: *Journal of Laryngology and Otology* 56:18-28 (Jan.) 1952.

The course and prognosis of this disease have been radically changed from a grave to a benign one since the advent of antibiotics. Operative injury or indirect infection from the teeth or antrum are the commonest etiological factors. Infection is more likely to gain a footing and to spread in the spongy alveolar bone than in the relatively compact bone which forms the wall of the antrum. The author has illustrated the blood supply from the internal maxillary artery and its anastomosing loops and explains on the basis of blood supply that the sequestra are likely to be limited or involve the whole bone, and that only in the fulminating cases is the infection likely to spread to neighboring bones. Orbital cellulitis and cavernous sinus thrombosis are due to direct spread.

The usual infecting organism is the staphylococcus aureus. Anaerobic streptococci are not uncommonly found.

The diagnosis is not difficult. The boring facial pain, pyrexia, swelling, trismus, nasal discharge, and the ocular symptoms suggest an osteomyelitis of the maxilla.

Cases often resolve with sequestration. Untreated, the disease may spread to other bones. Septicemia or intracranial complications are the cause of death. The treatment consists of penicillin, draining the pus, and then removing the sequestra.

There is a brief discussion of osteomyelitis in sucklings. Staphylococcus aureus is almost always the organism responsible. It seems to be generally agreed that the unerupted tooth germ is the commonest primary focus. It seems probable that the infection enters through an abrasion on the surface of the gum, and this theory gains support from the fact that in a large number of cases the mother is

found to be suffering from a cracked nipple, or some other staphylococcal infection of the breast at the same time. The infecting agent may also be the finger of a relative, nurse or doctor or the teat of a feeding bottle. A blood-borne infection is theoretically possible but is very much less likely.

Infection of the antrum is secondary only. The treatment is the same as for osteomyelitis in older children or adults. The paper is illustrated with case reports.

BOIES.

BRONCHI

Grasses as Foreign Bodies in the Bronchus and Lung.

Jackson, Chevalier: Laryngoscope 62:897-923 (Sept.) 1952.

Dr. Chevalier Jackson presents a very thorough and well documented dissertation on the rather uncommon problem of grasses as foreign bodies in the pulmonary tree. Seven cases observed in the Temple University Clinic and 28 cases gleaned from the literature are reported. These are divided, according to the genus of the grass inspissated, the pathological changes produced and the clinical progress of the case, into two distinct classes, viz.: the "lodging type" and the "one way travel" type.

The "lodging type" remained in the bronchus until removed bronchoscopically. The "one way travel" type of grass was driven through the lung and thoracic wall and was extruded through a fistula onto the skin surface.

A vivid description of the mechanism by which these grass spikes are forced into the periphery of the lung is presented with excellent explanatory illustrations.

In completing this excellent presentation, the initial symptoms, problem of differential diagnosis, bronchoscopic appearance, treatment and prognosis are ably presented. It is worthy of special emphasis that even in the cases where serious pulmonary infection and perforating fistula were present, there was rapid healing and an absence of mortality.

SENTURIA.

Results of Radiation Therapy for Bronchogenic Carcinoma. A Statistical Analysis of 125 Cases.

Adelman, Bernard P.: Radiology 59:390-398 (Sept.) 1952.

Statistical study of 125 cases of bronchogenic carcinoma treated by x-ray therapy fails to reveal any definite trend to increased longevity with increasing tumor dose. Tumor doses varied upward to 5000 roentgens. Average age of patient was 54 years, extremes 20 and 74 years. 89.8% were males. Average time from onset of symptoms to diagnosis was 6.5 months.

Physical factors of irradiation are discussed. All cases at present are treated by the protracted fractionation technic. Other factors such as age of patient at onset of disease, duration of symptoms prior to diagnosis and sex are discussed. Six charts and one table summarize the survival rates in relation to these factors and tumor dosage. There is some difference in survival following therapy for the different histologic types, the longest survival being expected with squamous-cell carcinoma. Forty-three references.

JORSTAD.

Radiologic Diagnosis of Respiratory Lesions in Children.

Chapman, Sam B.: Radiology 58:705-710 (May) 1952.

Some of the technical and diagnostic problems of pediatric radiologic practice are reviewed. Eight representative radiographs of congenital anomalies, foreign bodies, infectious processes, and neoplasma in the lungs are shown. The author emphasized that the radiologists must classify the lesions anatomically and pathologically, tread lightly where etiological diagnosis is concerned until he has follow-up studies and the collaboration of other diagnostic departments.

JORSTAD.

MISCELLANEOUS

Smoking and Lung Cancer.

Editorial: British Medical Journal pp. 1299-1301 (Dec. 13) 1952.

In England and Wales 13,233 deaths were registered from cancer of the bronchus of the lung in 1951. This exceeded the number of deaths from respiratory tuberculosis. Out of a random

sample of 400 men now aged about 55, at least 10 must be expected to die of lung cancer within the next 20 years in comparison with 200 from all other causes, even though the death rates remain at their previous level.

Reference is made to the first report of Dr. Richard Doll and Professor A. Bradford Hill published in the *Journal* in 1950. This inquiry was conducted among 2,475 patients in 20 London hospitals, leading to the conclusion that "smoking is a factor, and an important factor, in the production of carcinoma of the lung." Their results were confirmed in almost all aspects by a similar study of 684 cases of bronchogenic carcinoma by Wynder and Graham in the U.S.A. The editorial comments, "where do we go from here?" This inquiry has continued.

Their statistics show that the probability of a causative connection between an agent and a disease is so great that we are bound to take what preventive action we can, accepting the theory as though the proof were absolute until further research leads to some modification. It would seem that such a position has now been reached with lung carcinoma in that tobacco has been incriminated as the vehicle conveying an agent responsible for a large proportion of the cases. Intensive research on the chemical constituents of tobacco and tobacco-smoke is now needed, and it is surely incumbent upon the tobacco manufacturers to do this. In the meantime the younger generation will have to decide each for himself or herself whether the additional risk of contracting lung carcinoma is worth taking. For a middle-aged man lung cancer diminishes his expectation of living 20 years by about one-twentieth. The apportionment of responsibility for that due to smoking and other factors such as atmospheric pollution can be roughly estimated, since the statistical pieces of the puzzle now seem to fall into place fairly well and make sense. If the annual total of deaths of men from lung cancer is taken as 11,000, of whom 7 out of each 1,357 would be non-smokers, the total male non-smokers dying annually would be 56; and if the proportion of non-smokers in the population at the relevant ages is taken to be 5%, the remaining 95% would also be exposed to the same risks from causes other than tobacco—which gives a total of about 1,100 deaths of men due to these causes, compared with 10,000 attributable to tobacco. By similar reasoning, with an annual total of about 2,000 deaths among women, of whom 37 out of each 108 would be non-smokers, the total female non-smokers dying would be 685 and, taking 60% of the female population at the relevant ages to be non-smokers, we can estimate that the total lung-cancer deaths of women due to causes other than tobacco would be about

1,100, as for men, the other 900 being due to tobacco. This seems to dispose of the idea that there is a sex difference in susceptibility to lung cancer, the large excess of male mortality being explainable by greater use of tobacco among men.

BOIES.

A Study of the Etiology of Carcinoma of the Lung.

Doll, Richard, and Hill, A. Bradford: British Medical Journal pp. 1271-1286 (Dec. 13) 1952.

This is a report of a very complete investigation designed to throw light on the etiology of carcinoma of the lung. It involves 5,000 hospital patients who were interviewed in private hospitals over a four-year period and for a limited purpose in two rural areas. The questions asked covered a very wide range, including the occupational histories, where the patients lived, the forms of domestic heating in their homes, their previous attacks of respiratory illness, and their habits with regard to smoking.

Preliminary figures had been published in a previous paper by the same authors in 1950. The present paper gives corresponding data for the whole of the material collected. The main comparisons are between 1,465 patients with carcinoma of the lung and an equal number of "matched control" patients with other diseases, each of these being carefully chosen so as to be the same age, the same sex, and so far as possible in the same hospital at the same time as a lung-carcinoma patient.

No appreciable difference between the two groups in the matter of social class was found, and there was no association between any type of occupation and lung-carcinoma which would suggest the presence of an etiological agent likely to be of general significance.

With regard to the possible effects of fumes in the atmosphere, both within and without the home, there was no significant difference between the groups.

In conformity with the national death rates rather fewer of the lung-carcinoma patients lived, or had lived, in the countryside and more, correspondingly, in the towns.

The lung-carcinoma group in comparison with patients with other forms of cancer more often gave a history of a previous attack of pneumonia and of chronic bronchitis. Detailed analysis of the data suggests, however, that this difference may be due merely to

the lung-carcinoma patients, with their respiratory symptoms, recalling more readily than other persons their previous attacks of respiratory illness. The data are not accurate enough for an etiological relationship to be postulated.

Of the 1,357 men with carcinoma of the lung 7, or 0.5% were non-smokers; of the 108 women there were 40, or 37%. The corresponding figures for their paired controls were 61 men (4.5%) and 59 women (54.6%). Of the men with lung carcinoma 25% reported that they had been smoking, before the onset of their illness, an average of 25 or more cigarettes a day (or the equivalent in pipe tobacco). For women these proportions were 11.1% for the carcinoma group and 0.9% for the controls.

Estimated death rates for Greater London indicate that the mortality from carcinoma of the lung may increase in approximately simple proportion with the amount smoked. Amongst men of ages 45-64 the death rate in non-smokers is negligible, while in the heavier-smoking categories it is estimated to reach 3 to 5 deaths per annum per 1,000 living.

The validity of these various results is studied, and it is concluded that the association between smoking and carcinoma of the lung is real. It is not argued that tobacco smoke constributes to the development of all cases of the disease—a most unlikely event. It is not argued that it is the sole cause of the increased death rate of recent years nor that it can wholly explain the different mortality rates between town and country.

BOIES.

Diffuse Subdural Suppuration.

Wood, Philip H.: Journal of Laryngology and Otology 56:496-515 (Oct.) 1952.

The author reviews an experience with diffuse suppuration within the subdural space. He remarks that this is probably the least known of the intracranial infections that may complicate sinusitis or otitis media. He reports eleven personally treated cases. The introduction of penicillin has changed the entire course of this infection. The therapeutic use of systemic and local penicillin combined with prompt surgical drainage has brought apparent cures but has led to new problems in the matter of chronic smouldering infection.

The author advocates prompt drainage using multiple burrholes together with penicillin therapy. He discusses a new entity

of chronic subdural granuloma which is attributed to delayed or inadequate drainage. Seven of the author's cases recovered.

BOIES.

Abuse of Antibiotics.

Editorial: *British Medical Journal* pp. 1301-1302 (Dec. 13) 1952.

This editorial was inspired by a letter protesting against the "unthinking universal employment" of chemotherapeutic agents. Two cases were cited in one of which the prophylactic use and in the other the therapeutic use of antibiotics obscured the development of serious complications.

The prophylactic use of antibiotics is arguable as to whether in given circumstances it is indicated at all. It is a common practice to give penicillin before and after a clean operation for the dual purpose of protecting the operation site against accidental contamination and of preventing chest complications. It sometimes fails in the latter purpose and when it does, the infection developing is penicillin-resistant and consequently much more difficult to treat. It is evidently impossible to lay down hard and fast rules about this practice, although there are certain types of patients in whom it is clearly indicated. Penicillin cover is centainly necessary for dental extraction or tonsillectomy in a rheumatic subject. It is equally clearly indicated in operations in a septic field, where any interference may otherwise cause an acute exacerbation. This applies particularly to the surgery of infected bone, and the use not only of penicillin but of other antibiotics has greatly facilitated major operations on the lung. Operations on the alimentary tract are in a different class, since here only the normal flora is usually to be feared. The use of sulphonamides or antibiotics as intestinal antiseptics preparatory to operations on the colon is a sound and established practice, but it relies rather on the local than on any general effect. There is no similar procedure for operations on the stomach or esophagus, nor is there the same need for it, the flora of this area being much scantier and the factor of distension by gas inoperative. Whether systemic antibiotic prophylaxis is indicated in such operations may presumably depend on the features of the individual case.

As a cover for operations on the urinary tract a sulphonamide is indicated—unless some infection insensitive to these drugs already exists—antibiotics being held in reserve to deal with later developments, if any.

For most operations in healthy and bacteria-free tissues there should be no need for any such prophylaxis.

The letter that inspired this editorial brought a more general charge, particularly against the younger generation of doctors: that antibiotics are being used indiscriminately for trivial conditions. It is contended that they should not be used for trivial aliments, as mere febrifuges or as placebos. There are many reasons for such restraint, of which the more obvious are economy and the desirability of making a diagnosis before starting treatment, particularly when the treatment is apt to obscure it altogether. Also, there is the risk of sensitization. The proportion of patients sensitized to penicillin is rising slowly and steadily and if the treatment which produced this state was in fact unnecessary it is a pity that they should have to run serious risks on a later occasion when they really need it.

A more serious reason is acquired bacterial resistance, not so much in the individual as in the population as a whole. All the newer antibiotics are slowly wasting assets by reason of this change, but if they were restricted to their proper fields of use we could enjoy them for many years longer. Let anyone tempted to use one of them for, say, a common cold reflect that this type of use may abolish its effect in a patient with septicaemia at some time in the future.

BOIES.

Senile Maxillary Osteoneuritis. A New Clinical Entity. Preliminary Report.

Prujansky, Bunim: Oto-Laryngologica Israelinana, No. 2, Page 28, 1952.

The author believes that a new clinical entity called senile maxillary osteoneuritis should be recognized. It occurs in edentulous people about 60 years of age. Symptoms consist of sudden persistent cutting, boring, burning, stabbing pain in the region of the superior maxilla which may radiate into the temporal region and into the ear. Examination with the palpating finger reveals a tender area in the alveolar process of the superior maxilla. X-ray examination may show a translucent area corresponding to the tender spot. Histopathologic examination of tissue reveals resorbtion and necrosis of bone trabeculae, the development of new bone tissue, granulations and connective tissue.

It is believed that a latent post-dental focus may be activated by pressure of dentures and that as the inflammation spreads a neuritis develops. Bacterial examination of the focus reveals streptococcus

haemolyticus or non-haemolyticus and staphylococcus albus. Treatment is surgical and consists of incision of the mucosa and curettage of the inflamed soft bone. Two illustrative case histories are presented.

SENTURIA.

The Clinical Significance of Hemoptysis.

Sonders, C. R., and Smith, A. T.: New England Journal of Medicine 247:21 (Nov. 20) 1952, pp. 790-793.

This is an excellently presented study of a series of 105 patients whose primary complaint was bleeding from the lower respiratory tract. 30 cases, or 28.2% were found due to bronchiectasis; 19, or 18%, to chronic bronchitis; while in 19, or 18%, the cause could not be determined. Tuberculosis, carcinoma, hypertension, cardiac failure were among the other diagnoses made but none of these made up more than 5% of the total number.

Fourteen of the 19 cases, in which no cause could be determined, have had a follow-up period averaging 25 months (1 month to 11 years) and in none of these has any pulmonary disease developed.

As the examination of these cases included bronchoscopy and bronchography this observation is somewhat consoling, if not, elucidating, to the endoscopist who, at times is forced to report negative findings in this condition.

HILL.

Books Received

Clinical Allergy.

By French K. Hansel, M.D., Director Hansel Foundation for Education and Research in Allergy; Chief of Allergy Service, DePaul Hospital, St. Louis, pp. 1005, Royal 8 vo., illustrated, C. V. Mosby Company, St. Louis, 1953. (Price $17.50.

Today when many a man's textbook is co-authored by as many of his friends as can be induced to participate, it is a delight to pick up the new volume on Clinical Allergy, by French Hansel. Picking it up is a task in itself, for the book is large octavo and the pages are a thousand and five.

While encyclopedic in treatment, and omitting nothing of the slightest relationship to the subject, the work is presented, as only a single author can do it, in such a way as to make consecutive reading a pleasure.

To record even the chapter headings would require more space than is here available. There are 53 chapters dealing with everything from the basic concepts of anaphylaxis and a comprehensive glossary of the terminology to the most minute descriptions of allergic manifestations in various regions and tissues, the sinuses and asthma, allergic dermatitis, cerebral allergy and headaches. While the author contributes enough from his own wide experience to maintain coherence and guide the reader, this book is essentially an epitome of the vast literature on allergy.

Hearing Aids, Their Use, Care, and Repair.

By Matthew Mandl, Lecturer in Electronics and Television, Technical Institute, Temple University, Author of Mandl's Television Servicing, Co-author, Television and FM Antenna Guide, pp. 158, 8 vo, Illustrated, Macmillan Company, New York, 1953. (Price $3.50)

The Function of the Vestibular Organ.

By A. A. J. vanEgmond, J. J. Groen and L. B. W. Jongkees. 109 pages, illustrated. S. Karger Ltd., Basel, New York. (Price 14 Swiss francs)

Let's Hear It.

By George W. Frankel, M.D. 8vo, 64 pages. Stratford House, Inc., New York. (Price $1.00)

Notices

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

April 21-25, 1953, in New Orleans, La., at the Roosevelt Hotel.

October 6-10, 1953, in Chicago, Ill., at the Palmer House.

DEAN M. LIERLE, M.D., Secy.
University of Iowa
Iowa City, Iowa.

FIFTH INTERNATIONAL CONGRESS OF OTO-RHINO-LARYNGO-BRONCHO-ESOPHAGOLOGY

The International Committee for the International Congress of Oto-Rhino-Laryngology has invited the Dutch Oto-Rhino-Laryngological Society to organize the Fifth International Congress in the Netherlands.

This Congress will take place on June 8-15, 1953 in Amsterdam.

For further information please address W. H. Struben, J. J. Viottastraat 1, Amsterdam-Z.

ACADEMY HOME STUDY COURSES

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

PAN-AMERICAN CONGRESS

Fourth Pan-American Congress of Oto-rhino-laryngology and Broncho-esophagology, Mexico City, January 1954. Dr. Ricardo Tapia Acuna, President, Dr. Chevalier L. Jackson, Executive Secretary, 1901 Walnut St., Philadelphia 3, Pa.

OTO-NEURO-OPHTHALMOLOGY

The Eighteenth International Congress of Oto-Neuro-Ophthalmology will be held in Bologna (Italy) from the 3rd to the 7th of May 1953. General Secretary, Dr. Giuseppe Cristini, Clinica Oculistica-Policlinico, Bologna (Italy).

NEW YORK EYE AND EAR INFIRMARY

The Alumni Association of The New York Eye and Ear Infirmary, 218 Second Avenue, New York, will hold its annual scientific program April 20th through April 24th. The scientific sessions in Ophthalmology will be held on the 20th and 21st; in Otolaryngology on the 23rd and 24th at The New York Academy of Medicine.

Hunter H. Romaine, M.D., Secy.

ANNALS

The management of the ANNALS desires to buy, at $1.50 each, copies of the following numbers which are out of print:

March, 1940
December, 1941
March, 1949
March, 1950
March, 1951

UNIVERSITY OF ILLINOIS

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology, divided into two sections:

A. Basic Section, September 21 through 26, 1953, devoted to surgical anatomy and dissection of the head and neck, and histopathology of the ear, nose and throat, under the direction of Dr. M. F. Snitman.

B. Clinical Section, September 28 through October 3, 1953, consisting of lectures and panel discussions, with group participation of otolaryngological problems and current trends in medical and surgical management.

Registration will be limited. Application for attendance at one or both sections will be optional. For information write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

UNIVERSITY OF ILLINOIS

For the period of the National Emergency, the University of Illinois College of Medicine will offer a combined three year residency training program in Otolaryngology, which will include the basic course material, in fulfillment of Board requirements.

Residents will rotate through the Research and Educational Hospitals, the Illinois Eye and Ear Infirmary, the Hines Veterans Administration Hospital and the various affiliated institutions. The residency will be so flexible that should it be interrupted because of military service, the period of training may be resumed upon returning to civilian life.

Under this arrangement, no course fee is to be involved and in the case of most of the aforementioned institutions, a stipend is provided.

For further information, kindly address: Head of the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

PROGRAM

OF THE ANNUAL MEETING OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

April 26-27, 1953

Primary Plasma Cell Tumors of the Upper Air Passages, Clyde A. Heatly, M.D.

Mesenchyme of the Nose and Sinuses, Aubrey C. Rawlins, M.D.

Primary Amyloid Disease of the Larynx, Julius W. McCall, M.D.

The Laminagraph as an Aid in the Diagnosis of Diseases of the Larynx, Leroy A. Schall, M.D. and A. S. MacMillan, M.D. (by invitation).

Clinical Significance of Laryngeal Keratosis as a Pre-malignant Lesion, F. J. Putney, M.D. and John J. O'Keefe, M.D. (by invitation).

Carcinoma-in-situ of the Larynx, Alden H. Miller, M.D. and Russell Fisher, M.D. (by invitation).

Chordoma. Final Report and Re-evaluation of Treatment, Henry B. Orton, M.D.

Neurilemmona (Schwannoma) of the Larynx, L. Chester McHenry, M.D.

Hemilaryngectomy and Subtotal Laryngectomy with Immediate Skin Graft, Frederick A. Figi, M.D.

Treatment of Hyperplastic Sinusitis, Francis W. Davison, M.D.

Pharyngeal and Laryngeal Phenomena of Vascular Origin, French K. Hansel, M.D.

Precision Bronchology. A Must in Thoracic Surgery, Robert E. Priest, M.D.

Surgical Complications of Diverticulum of the Pharynx, Kenneth A. Phelps, M.D.

Realism in the Surgery of the Tonsils and Adenoids, Chas. D. Blassingame, M.D.

Recent Items on Anesthesia of Interest to Laryngologists, John Adriani, M.D. (by invitation).

(The above papers will be printed in the June Annals.)

PROGRAM

OF THE ANNUAL MEETING OF THE

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

April 28-30, 1953

Low Tone Deafness from Eighth Nerve Section, Harold F. Schuknecht, M.D. (by invitation) and Richard Woellner, B.A. (by invitation).

Bronchogenic Carcinoma: The Role of the Bronchoscopist in Its Early Diagnosis, Alton Ochsner, M.D. (by invitation).

The Formation of a Hearing and Speech Center, Gordon D. Hoople, M.D. and Louis M. DiCarlo, Ph.D. (by invitation).

The Role of Rhinoplasty in Rhinology, Maurice H. Cottle, M.D. (by invitation).

Laryngeal Stenosis—Corrective Plastic Procedure for Failures Following Bilateral Abductor Paralysis Surgery, DeGraaf Woodman, M.D.

Carcinoma of the Oral Cavity, Hans von Leden, M.D. (by invitation).

Anatomical and Functional Considerations in Temporal Bone Surgery, Julius Lempert, M.D.

Neurofibromas of the Larynx, Frederick A. Figi, M.D. and David B. Stark, M.D. (by invitation).

The Identification and Clinical Significance of Large Phagocytes in the Exudates of Acute Otitis Media and Mastoiditis, William T. K. Bryan, M.D.

Trans-Oral Arytenoidectomy for Bilateral Vocal Cord Paralysis, Fred Z. Havens, M.D.

The Selection of Hearing Aids as an Office Procedure, Werner Mueller, M.D.

Medical Problems, Louis H. Bauer, M.D., President of the American Medical Association (by invitation).

Some Newer Observations on Nasal Cilia, G. Edward Tremble, M.D.

A Re-Evaluation of Semon's Hypothesis, Louis H. Clerf, M.D. and William H. Baltzell, M.D. (by invitation).

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

(As of October 1, 1952)

Audiotone Model 11
Audicon Models 400 and 415
Audivox Model Super 67
Aurex Models L & M

Beltone Symphonette Model
Beltone Mono-Pac Model M

Cleartone Model 500
Cleartone Model 700
Cleartone Regency Model

Dahlberg Model D-1
Dahlberg Junior Model D-2
Dahlberg Model D-3
Dahlberg Model D-4
Dysonic Model 1

Electroear Model C

Gem Model V-35
Gem Model V-60
Goldentone Models 25, 69 & 97

Maico UE Atomeer
Maico Quiet Ear Models G & H
Maico Model J
Mears (Crystal and Magnetic) Aurophone Model 200
Micronic Model 303
Micronic Model "Mercury"
Micronic Star Model
Microtone Classic Model T-9
Microtone Model T-10
Microtone Model T612
Microtone Model 45

National Cub Model (C)
National Model D (Duplex)
National Standard Model (T)
National Star Model (S)
National Ultrathin Model 504
National Vanity Model 506
Normatone Model C

Otarion Model E-4
Otarion Models F-1, F-2 and F-3
Otarion Models G-2 and G-3

Paravox Model D "Top-Twin-Tone"
Paravox Model J (Tiny-Myte)
Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Radioear Permo-Magnetic (Multipower)
Radioear Permo-Magnetic (Uniphone)
Radioear All-Magnetic Model 55
Radioear Model 62 Starlet
Radioear Model 72
Radioear Model 82 (Zephyr)
Rochester Model R-1
Rochester Model R-2

Silvertone Model J-92
Silvertone Model P-15
Solo-Pak Model 99
Sonotone Model 900
Sonotone Models 910 and 920
Sonotone Model 925
Sonotone Model 940
Sonotone Model 966
Super-Fonic Hearing Aid

Televox Model E
Telex Model 97
Telex Model 99
Telex Model 200
Telex Model 300-B
Telex Model 400
Telex Model 500
Telex Model 952
Telex Model 1700
Tonamic Model 50
Tonemaster Model Royal
Tonemaster Cameo Model

Unex Midget Model 95
Unex Midget Model 110
Unex Models 200 and 230

Vacolite Models J and J-2

Western Electric Models 65 and 66

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)
Aurex Semi-Portable
Precision Table Hearing Aid
Sonotone Professional Table Set Model 50

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Blvd., Chicago 11, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14, Mass.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

American Broncho-Esophagological Association

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.

American Laryngological Association

President: Louis H. Clerf, 1530 Locust St., Philadelphia, Pa.
Secretary: Harry P. Schenck, 326 So. 19th St., Philadelphia, Pa.
Meeting: New Orleans, La., April 26 and 27, 1953.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis LeJeune, Prytania and Olive Sts., New Orleans, La.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: New Orleans, La., April 28-30, 1953.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

American Otological Society

President: Dr. Albert C. Furstenberg, University of Michigan Hospital, Ann Arbor, Mich.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago, Ill.
Meeting: New Orleans, La., May 1-2, 1952.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: Kenneth L. Craft, M.D., 23 East Ohio Street, Indianapolis, Indiana.
President-Elect: Albert D. Ruedemann, M.D., 1633 David Whitney Building, Detroit 26, Michigan.
Secretary-Treasurer: Michael H. Barone, M.D., 468 Delaware Avenue, Buffalo 2, New York.
Meeting: Chicago, Illinois on October 16, 1953.

International Bronchoesophagological Society

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Second International Congress of Bronchoesophagology, June 15th and 16th, 1953, Albert Plage, Le Zoute, Belgium.

Pacific Coast Oto-Ophthalmological Society

President: C. Allen Dickey, 450 Sutter St., San Francisco, Calif.
Secretary-Treasurer: Howard P. House, 1136 W. Sixth St., Los Angeles, Calif.
Meeting: Los Angeles, Calif., 1953.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo M. Alonso, Montevideo.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U.S.A.
Meeting: Fourth Congress of Oto-rhino-laryngology and Broncho-esophagology.
President: Dr. Ricardo Tapia Acuña, Mexico City.
General Secretary: Dr. Maximo García Castaneda.
Time and Place: February 28th to March 4th, 1954, Mexico City.